EXHIBIT 5

Report of Edward J. Calabrese, Ph.D.

Sullivan, et al. v. Saint-Gobain Performance Plastics Co., No. 5:16-cv-000125-GWC (D. Vt.)

PROFESSIONAL BACKGROUND

I am a tenured professor of toxicology within the Department of Environmental Health Sciences, School of Public Health and Health Sciences of the University of Massachusetts at Amherst. I have been on the faculty at the University of Massachusetts since 1976 as an assistant professor, then promoted to associate professor in 1980 and to full professor in 1982. I previously was an assistant professor of environmental and occupational medicine from 1974-1976 at the University of Illinois School of Public Health in Chicago. While at the University of Illinois I was the Assistant Director of the Environmental Research Resource Center, a very active entity that focused on assessing key environmental health issues for the state, including lead exposure, ambient ozone, sulphur dioxide and nitrogen dioxide, radium in drinking water, effects of coal gasification, amongst other issues facing the state at that time.

I have had an active research program throughout my career at the University level with about 45 years of uninterrupted external funding. I have published over 835 papers in the peer-reviewed literature, authored 12 books on toxicology and risk assessment and edited over two dozen books. I have numerous papers that have been extensively cited in the Web of Science data base with more than 30 papers being cited more than 100 times and with an H-index of 54 (i.e. 54 papers being cited at least 54 times). These scores are far higher than average within the scientific community. I have given over 750 invited lectures, seminars and conferences presentations in many countries. A copy of my curriculum vitae is attached as Exhibit A.

The research areas over my professional life have focused on the occurrence, causes and mechanisms of human inter-individual variation (i.e., age, genetics, diet, gender, pre-existing disease conditions) (i.e., why some people get sick and others don't even though similarly exposed); how to extrapolate from animal models to humans-opportunities and limitations; the occurrence, nature and mechanisms of adaptive responses to low doses of toxic agents and their evolutionary origins and public health applications; the nature of the dose response in the low dose zone; and the historical foundations of toxicology, and cancer and non-cancer risk assessment within the context of the dose response.

I have also had several long-term research collaborations with University of Massachusetts professors of epidemiology, which have resulted in significant contributions to the scientific literature. These areas include the effects of sodium in drinking water on human health (e.g., blood pressure in children), the effects of working in the clean work of semi-conductor chip manufacture on reproductive health, and the estimation of how much soil children and adults ingest and how this relates to human risk assessment. In the case of the semi-conductor research, it lead to removal of toxic agents from chip manufacture and their replacement with less toxic substances. This was a major development in the field and lead to widespread national debate and publicity.

In the case of the soil ingestion, our findings have provided the principal basis for the EPA soil ingestion estimates in their Exposure Factors Handbook over multiple editions to the present. Our research has provided the scientific basis for soil remediation standards for hazardous wastes sites/Superfund clean-up actions.

These research achievements have led to my being awarded several honors such as the International Marie Curie Prize and an Honorary Doctorate from McMaster University, a leading

educational and research University in Canada. I have also received the highest awards from two professional scientific societies even though I am not a member of these societies.

I have served in numerous capacities as an advisor to state and federal agencies as well as to international organizations, such as the NATO countries safe drinking water committee. I have served on multiple United States (US) National Academy of Sciences committees, including several of the Safe Drinking Water Committees and served as one of the lead advisers to this committee.

I have also served on the Air Cabin Safety Committee that recommended the end of smoking on airplanes. This recommendation lead to the elimination of smoking during air travel throughout the US. I also was a member of the Food and Nutrition Board of the Institute of Medicine that has been responsible recommending daily acceptable levels of vitamins and minerals for human consumption.

I was also a member of the Board of Scientific Counselors for the Agency for Toxic Substances and Diseases Registries (ATSDR) for five years. I helped to create the Northeast Regional Environmental Public Health Center at the University of Massachusetts, which was designed to enhance communication and cooperation amongst the State Departments of Public on environmental pollution issues and to also enhance interactions with the various State Departments of Environmental Protection and Agriculture with regional and national EPA.

During this time period I also helped to create one of the longest running academic conferences in the country. Starting in 1986, I have co-directed a national conference on Soil, Sediment and Water Contamination. This ongoing conference attracts about 900 people each year, from about 40 countries. It provides significant scientific leadership and education in this area.

Within this context I helped to create the now longstanding peer-reviewed journal Soil and Sediments. I have also co-directed a similar conference on the west coast since 1990, which is usually held in San Diego. I have also created and directed an annual conference on the biological effects of low doses of environmental and pharmaceutical agents on human health. This lead to the creation of the journal Dose Response, which I have edited for over 15 years. I have also helped to create and been the first editor of the journal Human and Ecological Risk Assessment, a position I held for nearly a decade prior to becoming the editor of Dose Response.

In addition to my editorial duties, I also have long served as a peer reviewer for toxicological and risk assessment articles for multiple journals and have done so for several decades. I typically review approximately one paper per month for journals.

I have also created the only database on agents that can induce cancer with a single exposure. Due to concerns with unexpected exposure/releases of toxic agents, I have been invited to make multiple presentations to various National Academy of Sciences committees on the topic of single exposure carcinogens and/or unanticipated toxic substances release.

Of further note is that I was the principal consultant to the state of Colorado in their prolonged litigation concerning the Rocky Mountain Arsenal. This activity, which occurred over the 1988-2002 time period, lead to the successful resolution of this highly contentious area involving both human and ecological risk assessment and the creation of the Rocky Mountain Arsenal Refuge Center, a 27 square mile tract of land for wildlife and human visitors.

In summary, I have had an extensive and active career in the field of toxicology and risk assessment, with in depth and broad research activities with significant accomplishments. I have also had much experience in the advising of governmental agencies at the highest levels and in the

evaluation of all types of scientific papers. Based on this background, I am qualified to evaluate the reliability of toxicologically based assessments that affect human health and to provide guidance on how to conduct causal analyses for environmental induced human disease conditions.

OPINIONS OF EDWARD CALABRESE

My report is divided into two corresponding sections. The first section provides a critical conceptual overview of causal assessment, with application to the analysis of Dr. Ducatman in his two reports (i.e., the September 1, 2017 class certification report and the December 15, 2017 merits report). The second section addresses the specific studies and opinions of Dr. Ducatman as contained in his two reports.

SECTION 1: GENERAL PRINCIPLES AND EVALUATIONS

Assessing Causation from Environmental Exposures in Humans

Causation determinations in humans are very challenging especially when exposures are generally in the low dose zone, when the endpoints at issue are commonly observed in unexposed people such as, for example, elevated cholesterol, hyperlipidemia (i.e., abnormally high levels of any or all lipids or lipoprotein in blood), and elevated uric acid levels, when the exposure histories to agents of concern and confounding agents are very uncertain, as well as when the endpoints that are measured are highly variable both between and within individuals and over time. The causality issue will also be further challenged by concerns associated with human genetic variability, and the difficulties of trying to estimate exposures to dietary, environmental, and medicinal drugs that

affect the endpoints at issue over periods that preceded birth and after birth but for which there is little or no documentation or memory.

These complexities for assessing human risks to exposures to toxic and/or carcinogenic substances are seen within the general evaluative frameworks for most agents. The above variables and others are part of the typical human condition and make it difficult to confidently determine causality when the substances at issue are present in low concentrations and reflect highly diverse patterns of exposure that may markedly change by day, week, season, and year. Even in the most studied subjects, such as with ionizing radiation, where many thousand animal model studies exist and a voluminous human epidemiological literature also exits, there remain recognized and generally accepted uncertainties in which risks from low level exposures cannot be discerned from background. In these cases, any risks are estimated by extrapolation procedures based on biostatistical models that cannot be verified or validated. In these cases, such estimates are based on assumptions or belief systems.

It has been possible in the past to estimate with confidence the effects of very high levels of toxic substances, such as smoking a pack or more of cigarettes per day for several decades, because they had the potential to induce adverse effects and to overwhelm biological variability and adaptive responses. However, in the modern era of widespread low dose exposures, discerning causality is very challenging. For example, while estimating the effects of 20-40 cigarettes per day for several decades has been possible with epidemiological methods, such methods would be severely challenged to estimate adverse biological effects if the exposures to be assessed were only 1-2 cigarettes per week for two decades. That is the challenge of human population studies in the low dose zone. Would such exposures actually induce adverse effects and, if so, could they be

reliably identified in the human population, given other competing causes of illness and death, as well as issues with population migrations from one city or state, amongst other factors.

Background variability for common conditions has long been recognized and it creates uncertainty in causality assessment. It is therefore necessary for adverse effects to be sufficiently elevated and exceed those occurring as a result of chance. It is also necessary that study findings be assessed for bias and confounding and be repeatedly replicated using study designs that can reliably detect possible causal effects. (See Ducatman deposition, pages 18-20.) In the case of the studies cited within Dr. Ducatman's reports, the overwhelming majority used a cross-sectional design that lacks a temporal component, preventing causality inferences. Such studies can be useful for generating hypotheses, but not identifying causes.

On page 37 of his deposition, Dr. Ducatman acknowledged that his opinions as expressed in both PFOA reports were prepared without Plaintiff–specific data. On page 39, he likewise acknowledged having no Plaintiff specific information on the extent to which such subjects ingested tap and/or bottled water. Thus, on a key exposure parameter for PFOA risk assessment for Plaintiffs in Bennington and North Bennington, Vermont, the opinions lacked necessary exposure data, an essential component of the risk assessment process when exposure from tap water is the question. Despite this statement on page 13 of the class certification report, Dr. Ducatman stated that the "Bennington population [was] homogenous only in their exposure to PFOA through their drinking water." This is a statement that is based on an unfounded assumption. However, opinions need to be empirically based, not assumptions (see Ducatman deposition, page 11).

PFOA and Peroxisome Proliferator Receptors (PPAR)

In the case of PFOA, one of the principal mechanisms of action in high dose experimental animal studies is believed to involve the activation and interaction of probably two peroxisome proliferator receptor (PPAR) types (PPAR-alpha and PPAR-gamma). The activation of the PPAR alpha receptor can vary markedly between species. In the case of experimental animal toxicological models, the principal animal strains used are much more sensitive to the activation of the alpha receptor than humans. This is a key mechanism that can account for the heightened susceptibility of these animal models to PFOA and their lack of capacity to predict human responses.

In humans, the alpha receptor is principally expressed in tissues that have high rates of fatty acid metabolism. The alpha receptor, among other things, targets genes that direct the process of lipid metabolism and guide the regulation of increasing HDL (i.e., the "good" cholesterol) and reducing plasma triglycerides levels (i.e., a generally harmful lipid at high concentrations). The pharmaceutical and medical communities have developed various drugs to lower lipid levels in patients via the activation of the alpha receptor. In addition, many naturally occurring substances in the diet also can activate the alpha receptor, usually resulting in a decreased lipid profile and decreased markers of inflammation.

Moreover, the human diet and pharmacy provides copious possibilities of highly variable exposures to agents that act on these same receptors, causing a broad range of biological effects, many of which are beneficial, with numerous drugs designed to act on these receptors, even with the potential to modulate some of the purported effects listed by Dr. Ducatman. These exposures to naturally occurring peroxisome proliferation agents have occurred in each person since they were in utero. Yet these exposures are essentially never documented, quantified and evaluated

within the studies cited by Dr. Ducatman or by his own analyses. This is an important limitation as it prevents the capacity to properly assess the effects of such agents and the PFOA on biological systems.

The range of genetic variability with respect to the peroxisome proliferator receptors is substantial within human populations, with significant impact on health. However, this issue was not addressed and studied in environmentally oriented epidemiological studies.

These examples illustrate the challenges and uncertainties inherent in the conduct of low dose epidemiology studies and the efforts to discern any causal associations for PFOA. Nevertheless, the September 1, 2017 initial expert report and the December 15, 2017 merits report of Dr. Ducatman fail to address these concerns and data limitations.

In most of the epidemiological reports that Dr. Ducatman relies upon, the authors of such research papers emphasized study design weaknesses, such as the use of cross-sectional studies, which precluded the capacity to make causal inferences. In fact, these studies, which could not make causal inference due to study design limits, also failed to consider the issues noted above concerning dietary and medicinal exposures to other peroxisome proliferation inducing agents and key genetic variables affecting the activation of the peroxisome proliferation receptor. These factors are important in a causal assessment.

Dr. Ducatman's Failure to Incorporate Dose Response

The issue of dose response is a central factor in assessing causality and risks. As noted above, it is necessary to identify and quantify exposure to agents that may act through the same mechanism. Exposure to the numerous other peroxisome proliferator agents to which people are exposed was not done by Dr. Ducatman nor was it presented in any of his cited references. In

other chemical exposure cases dealing with complex mixtures, such as polycyclic aromatic hydrocarbons (PAHs), it has been assumed that they may act via the same mechanism. This has lead researchers to develop something called the Toxic Equivalency Factor (TEF). This TEF approach offers a biologically based way to summate or add the total exposure to agents that presumably act through the same receptor, after adjustment for the degree of receptor affinity for each chemical agent.

Unless one can quantify the extent to which the key receptors are activated by the various PFOA exposures and then differentiate these exposures from exposures to the similarly acting agents in the diet, then quantification of exposure for risk assessment purposes cannot be reliably done. This is another key failing of Dr. Ducatman's reports and in the papers he relies upon.

Dr. Ducatman's reports also failed to the address the issue of the nature of the dose response of PFOA. This includes how data at relatively high doses may be extrapolated to lower doses and any uncertainties in that extrapolation process and the magnitude of such uncertainties. In the PFOA literature, there is considerable variation with respect to the reported dose response relationships even in the low dose zone.

Numerous examples (Boudreau et al., 2003; Coperchini et al., 2015; Florentin et al., 2011; Hagenaars et al., 2011; Henry and Fair, 2011; Liu et al., 2017; Midgett et al., 2014; Rosenmai et al., 2016; Wan et al., 2014; Wirth et al., 2013; Yao et al., 2014) exist showing no biological effects over very wide ranges of exposure, while many other experimental studies show evidence of biphasic dose responses (i.e., where a low dose may produce a different, and even possibly beneficial effect and a high dose can produce a toxic effect) (e.g., Buhrke et al., 2015). These findings are typically reported with either whole animal or cellular systems and suggest that similar types of variation may occur within humans. Of course, all substances, even those that most people

would consider beneficial or even necessary for human life, such as water, salt, and oxygen, can be toxic at sufficient doses. (See Ductaman deposition, pages 28-29.) Yet Dr. Ducatman's reports fail to address the dose response issue at the level of exposure and response, precluding the capacity to offer any scientifically reliable opinions.

Dr. Ducatman Failed to Address the Limitations and Uncertainties in Extrapolating Animal Model Findings to Human Responses

Dr. Ducatman's reports make use of animal and cellular toxicology findings to try to support his presentation of epidemiological studies. These reports fail to consider the possibility that both qualitative and quantitative differences exist amongst mouse strains and amongst rat strains in their responses to PFOA. Not only is it very difficult to predict responses from one mouse strain to another and for one rat strain to another, but it is therefore even more difficult to predict a rat response based on mouse data. (See Ducatman deposition, pages 33-34.) Given such inter-strain and species responses, it follows that there is considerable uncertainty in extrapolating experimental animal model data to humans, often precluding meaningful quantitative understandings. (See Ducatman deposition, pages 31-32.) For example, it is generally accepted that if a chemical causes cancer in an animal model there is very little confidence that this agent would predict the degree of susceptibility in the human or even the location (i.e., organ) possibly affected. Such uncertainties are due to fundamental biological differences between humans and rodents as well as the grossly high doses used in the animal studies, which provide little insight into exposures at far lower doses potentially experienced by humans. The use of animal studies are therefore problematic with respect to their lack of qualitative and quantitative similarity to humans and to the striking lack of relevancy of the testing protocol for application to normal human exposures.

For instance, in the case of the widely used B6C3F1 mouse, it has commonly developed chemical carcinogen induced liver cancer, a relatively uncommon cancer from all causes in humans. Thus, the occurrence of chemically induced liver cancer in the B6C3F1 mouse grossly overestimates possible humans risks. For example, Carlborg (1979) reported that the EPA has estimated via the linear no threshold (LNT) single-hit model from data with the B6C3F1 mouse that current exposures to DDT, dieldrin, and aflatoxin were responsible for 153,000 liver cancers per year in the U.S. (page 565, Calabrese, 1983). However, there were only about 7000-8000 liver cancers per year in the entire U.S. from all carcinogens and tumor promoters.

It was widely recognized that the PPAR-alpha receptor for PFOA is much more easily activated in standard rodent strains than in humans subjects resulting in estimated potential risks where no risks may exist (Corton et al., 2018). Furthermore, human subjects display genetic polymorphisms of peroxisome proliferators, making animal extrapolation less precise and uncertain with respect to potential relevance to human responses (Contreras et al., 2013) (page 439, abstract). There are numerous other interspecies (e.g., metabolism) variables that could affect potential susceptibility to PFOAs in addition to the activation of the PPAR-alpha receptor. These are also important issues not addressed by Dr. Ducatman.

SECTION 2: SPECIFIC CRITIQUE OF DR. DUCATMAN'S OPINIONS

This section assesses the toxicity claims of Dr. Ducatman concerning the exposure of residents of Bennington and North Bennington, Vermont to PFOA from drinking water. A sample of nearly 500 people revealed a geometric mean blood concentration of 10.0 ug/L. Dr. Ducatman's reports argue that, compared to current U.S. population background levels, the relatively elevated

exposure to PFOA has/will likely enhance risks for a plethora of medical conditions. As a result of such exposures, Dr. Ducatman recommends that the individuals have long-term (30 years) medical surveillance to identify possible health-related issues before they may become significant.

Yet his reports are strikingly devoid of a technical/scientific analysis and appropriate rigor. They thus provide the reader with no sound or reliable basis for any finding of causality. Such a lack of scientific analysis is present in both of his reports. These glaring failures of analysis result in these papers not achieving even a minimal professional standard in the field.

Assessment of the Bases of Dr. Ducatman's Reports

Summary of Opinions

Dr. Ducatman's reports lack technical assessment, analysis, and methodology for how he arrived at his numerous opinions. As a result, the reports fail to provide documentable scientific support for his opinions.

In scientific writings, it is necessary that the bases and reasoning for one's opinions be presented. (See Ducatman deposition, page 10.) Yet the bases and reasoning were absent from both of Dr. Ducatman's reports for each and every opinion and outcome.

As a result of such fundamental limitations, the entire spectrum of Dr. Ducatman's opinions on diseases, purported adverse effects, causality, and recommendations for medical monitoring lack scientific justification and cannot be accepted. These criticisms will be highlighted, by way of example, for certain of the endpoints discussed in his two reports.

Methodological Limitations of Studies Relied Upon by Dr. Ducatman by Endpoint

Cancer

On page four of his merits report, under the heading "PFOA Exposure and Cancer," Dr. Ducatman states: "Cancer outcomes of PFOA exposure that are supported in the medical literature including kidney and testicular cancer" and lists three citations. On page five of his class certification report, Dr. Ducatman similarly lists as purportedly "Consistently established in multiple venues … Urogenital cancers including kidney and testicular cancer" with the same three citations. Yet his reports offer no discussion, information, or analysis concerning fundamental relevant inquiries a scientist would make when evaluating peer-reviewed literature, such as: (i) the route and (ii) duration of exposure, (iii) the dates over which the exposures occurred, (iv) the type of epidemiological study (e.g., cohort, case-control, cross-sectional), (v) the number of subjects, (vi) number of cancer cases, (vi) how the tumors were verified, (vii) statistical evaluation strategies/analysis, (viii) type and significance of possible confounding factors or bias, and (ix) other important considerations, such as the consistency or inconsistency of such findings across studies in different populations.

The examples just presented for kidney and testicular cancers were not isolated cases of failure to report professional evaluation on fundamental aspects and details of the cited studies. The failures extend to the manner in which Dr. Ducatman describes all studies upon which his opinions are based independent of endpoint.

Dr. Ducatman's opinions, which presumably include human epidemiological studies as their bases, fail to assess and integrate the reported findings using standard and broadly accepted methods. For example, a scientifically rigorous evaluative methodological approach, such as the

proper application of the Bradford Hill criteria, which is one method used to assess possible causality from statistically significant associations reported in epidemiological studies, would be necessary. However, Dr. Ducatman's reports do not cite, use, or apply a fundamental evaluative procedure or methodology to derive his opinions or put them into scientific context.

Dr. Ducatman's reports are devoid of a scientific methodology for assessing causation. These omissions, and others, represent serious methodological failings as they impair the reader's ability to evaluate and then determine the objectivity and validity of the offered opinions. Dr. Ducatman's reports are thus comprised of opinions that lack a rigorous or reliable scientific method and basis.

For example, in the "PFOA Exposure and Cancer" section of his merits report, Dr. Ducatman uses highly unusual qualitative terms that seem to represent his opinions concerning the strength of the findings, and possible causal relationships. More specifically, on page 4 he writes:

> (1) "The cancer outcomes of PFOA that are supported in the medicine literature are…."
>
> (2) "there is an indication of excess risk of prostate cancer"
>
> (3) a risk "was detected again when the PFOA exposure has been above the population median levels" and
>
> (4) "There is also an early indication of increased breast cancer risk."

The use of terms such as "supported," "indication," and "detected" by Dr. Ducatman lack scientific precision and fail to provide quantitative and causal meaning to the biological events (e.g., adverse effects) purportedly being evaluated.

Dr. Ducatman similarly uses unusual terms on pages five and six of his class certification report:

(1) "Consistently established in multiple venues"

(2) "Probable excess risks needing additional investigation"

On page 86 of his deposition, Dr. Ducatman testified that the phrase "consistently established" does not have any recognized or generally accepted definition in the medical and scientific community that he is aware of. He nonetheless advances it in a categorical heading in his class certification report.

In his reports, the terms listed above are functionally decoupled from the concept of dose response, statistical significance, and other necessary standard evaluative components of scientific evaluative processes.

These sections of Dr. Ducatman's reports on PFOA and cancer cannot be used to scientifically assess cancer causality or cancer risk. They thus offer no scientific foundation to support his opinion concerning the need for a health/medical screening program.

Dr. Ducatman also attempts to complement and support the cited epidemiological studies for kidney and testicular cancers with limited animal research findings. His analysis lacks the necessary specificity in this area as well. For example, even though the animal research is cited in an attempt to support the human cancer data, he presents no evidence that PFOA causes kidney cancer in animal models. Among other things, he fails to provide any documentation concerning (i) the number of relevant animal cancer bioassays, (ii) the specific animal models used (iii) their extrapolative relevance, (iv) the dose ranges employed, and (v) how this information would relate to human exposures. The report acknowledges that PFOA is not a renal mutagen, an observation that would significantly lower the possibility of PFOA being a bona fide carcinogen, based on the somatic mutation theory that is widely accepted, including by federal regulatory agencies when

performing cancer causation analyses. In summary, the use of experimental animal study data to support the inadequately documented epidemiology cancer studies is without an appropriate toxicological foundation or methodology.

Even toxicological references concerning oxidative stress in the presence of PFOA exposure cited by Dr. Ducatman lack specificity for animal model, cell line, dose, dose-rate, and its relationship to human exposures. This approach by Dr. Ducatman, therefore, does not provide support for an opinion asserting PFOA cancer causality, nor does it support his opinion concerning the need for a medical monitoring program.

In a similar fashion, Dr. Ducatman attempts to provide evidence of a possible mechanism or toxicological basis to support his earlier opinion of an association of PFOA with testicular cancer. However, consistent with his earlier approach, there is no scientific framework, foundation, or methodology provided that permits a meaningful evaluation and scientifically based opinion.

In fact, the studies cited by Dr. Ducatman display highly uncertain extrapolative relevance for humans. For example, Dr. Ducatman cites the work of Dankers et al. (2013) with a mouse tumor cell line. These authors note that they were uncertain of the potential relevance of their experimental animal model to predict human responses (page 389, right column). They highlighted important ways in which human male reproductive functions (e.g., sex steroid production, secretion) differ from both rats and mice (page 389, right column). They noted that such differences are mechanistically important and should be taken into account when attempting to translate effects found in animal models for human risk assessment (page 389, right column). However, Dr. Ducatman does not acknowledge such challenges and limitations as reported by the authors when extrapolating from rodent models to humans for PFOA testicular effects.

Dr. Ducatman also fails to discuss the issue of PFOA dosing in the whole animal (i.e., in vivo) studies. For example, in his testicular cancer section, such studies employ a gavage administration method of exposure, providing the entire day's exposure at one time, essentially pouring it down the experimental animal's throat. In contrast, consumption of PFOA in water by Vermont residents would involve repetitious consumption throughout the awake cycle of the day. Thus, there is a very significant difference in dose-rate (i.e., exposure all at once versus the total amount spread out over time).

Dose-rate has the potential to be a significant factor affecting how the agent may affect the body. The significance of dose-rate in toxicology and in assessing risk became highlighted as early as 1958 when William L. Russell reported dose-rate was a significant factor affecting the capacity of ionizing radiation to cause mutation in spermatogonia and oocytes of mice (Russell et al., 1958) (page 1550, right column). This finding would become greatly expanded and incorporated into the process of assessing risk. Yet Dr. Ducatman's reports fail to acknowledge this factor in his analysis.

Furthermore, with respect to the cited toxicological study by Mashayekhi et al. (2015), which assessed the effects of PFOA on isolated suspensions of rat liver and brain mitochondria, Dr. Ducatman offers no evaluation or method of how to interpret and extrapolate such artificial and experimental conditions to intact cells within a living organism. Dr. Ducatman also fails to provide a toxicological basis to assess how to relate a concentration of PFOA in a mitochondrial suspension without the presence of other cellular organelles and contributory adaptive processes.

Endocrine Disruption

On page five of his class certification report, Dr. Ducatman lists endocrine disruption as purportedly being "Consistently established in multiple venues." In his section on endocrine disruption chemicals in his merits report, Dr. Ducatman states that the human data of Bjerregaard-Olesen et al. (2016) and La Rocca et al. (2015) provided strong evidence for PFOA being an endocrine disruptor for sex hormones. However, in their discussion, La Rocca et al. (2015) stated that "we found no association between PFOA and PFOS for both blood and semen levels and infertility." (page 12439). The Liu et al. (2015) study cited by Dr. Ducatman also notes that in "a study of 256 individuals the PFOA was not associated with sperm concentrations and sperm motility." (page 1, right column). Dr. Ducatman's failure to reveal such negative findings is consistent with his pattern of failing to consider or address alternative hypotheses or contradictory data in the studies he cites.

Within this same endocrine disruption section, Dr. Ducatman highlights research of Buhrke et al. (2015) with liver cells. But Dr. Ducatman fails to note that the PFOA concentration used in that study was extremely high and, according to the authors, of "not of physiological relevance." (page 60, left column). In fact, the authors stated that the concentrations used in the in vitro studies (which are studies that take place in laboratory vessels, outside of living organisms) exceeded average population values by three orders of magnitude (page 106, left column). Thus, these findings that Dr. Ducatman used to try to support his opinion cannot be validly used to do so.

Dr. Ducatman also cites the findings of Halsne et al. (2016) concerning the effects of PFOA on MCF 10A cells. Dr. Ducatman again fails to place the findings of this research in proper context. For example, it would have been necessary to know that Halsne et al. (2016) stated that the MCF 10A model has limitations such that these cells may not be particularly relevant to

humans. That is, these cells appear to be a differentiated cell type and are either not present or only rarely present in normal mammary tissue in vivo. Based on this information, Halsne et al. (2016) concluded that "care should be taken to directly link our in vitro observations using PFAAs to specific mammary developmental events in vivo." (page 106, right column).

Dr. Ducatman also cites the findings of Sonthithai et al. (2016) in the endocrine disruption study with T47D human breast cancer cells in vitro. Dr. Ducatman neglects to point out that PFOA did not affect the activity of the estrogen response element over a concentration of 10^{-12} to 10^{-4}M, some eight orders of magnitude of concentration (i.e., over 100-million fold concentration range) (page 794, Figure 1a; page 795, Figure 3a). This is an enormous concentration range. It shows no biological effects or possible risks.

Thus, each of the references used by Dr. Ducatman to try to support his claim for cancer and endocrine disruption was inadequately assessed. Those assessments, and others by Dr. Ducatman, lack a reliable evaluative methodology and proper technical presentation – transparent or otherwise – and ignore the significant failings and limitations of those references that he chooses to cite. This methodologic failure is a characteristic of multiple sections of both of his reports.

Pregnancy

On page six of his class certification report, Dr. Ducatman lists pregnancy-induced hypertension as a "probable excess risk needing additional investigation" and cites one study. In his merits report, Dr. Ducatman presents an association between PFOA exposure and the incidence of pregnancy-induced hypertension, citing a series of papers in an attempt to support his opinions regarding this relationship. However, he fails to present the complexity and inconsistencies of the reported findings and this relationship.

Savitz et al. (2012) noted that "Preeclampsia was weakly associated with PFOA exposures in other analyses of this population (Savitz et al., 2012; Stein et al., 2009)." (page 1205, left column). In their follow up study based on birth records, they "found no consistent evidence of an association between estimated PFOA exposure and stillbirth, pregnancy-induced hypertension, pre-term birth or indices of fetal growth." (page 1201, abstract). These authors mentioned that pregnancy-induced hypertension is the endpoint that is most susceptible to inconsistencies of definition and inaccurate reporting. They also noted the problems of misclassification of both self-reporting preeclampsia and birth certificate codes for pregnancy-induced hypertension. Yet Dr. Ducatman provides no assessment methodology to evaluate such epidemiologically-based uncertainties and no discussion of the differences that exist amongst available studies. This methodologic failure is a characteristic of multiple sections of both of his reports.

Thyroid

In his section on thyroid effects in his merits report, Dr. Ducatman notes that several papers reported that PFOA altered thyroid hormones, including during pregnancy citing Webster et al. (2014), Yang et al. (2016), and Lopez-Espinosa et al. (2012). Yet he did not provide any description relating to the study methodology, nor any assessment of the strengths or weaknesses of these findings. Again, this methodologic failure is a characteristic of multiple sections of both of his reports.

In Webster et al. (2014), no information was provided regarding how the subjects were recruited. The reader was referred to an earlier Webster et al. publication. While this reference provided the basis for the sample population recruitment, Dr. Ducatman does not acknowledge that the authors reported sample selection bias. The authors noted that such sample selection bias affects the generalizability of the study to be applied reliably to a broader and/or different

population (i.e., external validity) (page 345, left column). Also not mentioned by Dr. Ducatman was that the key target comparison group biomarker for autoimmune disease in that study involved only 14 subjects. In fact, due to this limitation, the authors specifically stated that "our results should be interpreted with caution because of small sample size…." (page 345, left column).

The Yang et al. (2016) study involved a population from the Beijing, China general population, an urban environmental setting very different than rural Vermont. The authors in fact emphasized this very concept and concern about generalizability in their paper (page 6). They stated that thyroid hormone levels are often conflicting in different populations due to many variables such as regional differences, race, community development, industrial pollution, lifestyle, amongst other factors (page 6). Thus, the relevance of the Beijing findings to the Vermont population is uncertain, at best. Indeed, no firm conclusions were in fact drawn by the authors of this Chinese study.

The third study cited by Dr. Ducatman is that of Lopez-Espinosa et al. (2012). While the sample population represents a large number of children aged 1 to 17 in the mid-Ohio Valley, the authors noted that the cross-sectional nature of this investigation should be considered as "a major limitation because the sample measurements precluded determination of the time sequence between PFOA exposure and outcome." (page 1040, 3rd column to the right). This "major limitation" impedes the capacity of the study to provide causality assessments. The authors also cited other relevant limitations such as the use of recall for thyroid diagnosis and the failure to obtain measurements of several key thyroid hormone levels, which would have provided important insights as to the child's thyroid functions (page 1040, bottom right column). Other cited studies in the thyroid section of Dr. Ducatman's reports emphasize the failure of cross-sectional study designs to permit causality inferences, while others also noted the limitation of having very low

sample sizes. Yet Dr. Ducatman's reports fail to discuss these factors, even though they are regularly acknowledged and addressed by those in the field.

Other types of limitations are also noted, such as the Melzer et al. (2010) study, which based PFOA measurement on a single serum sample. Some of these studies documented the presence of nearly two dozen other PFAA agents in the blood of mothers further complicating causality assessments (Webster et al 2014). Subject samples were also assessed for the presence of other possible thyroid affecting agents such as polybrominated diphenyl ethers (PBbEs), PCBs, and organochlorine pesticides (page 340, right column). How, or the extent to which, such concomitant exposures and other complex variables may affect data evaluation and interpretations was not addressed by Dr. Ducatman.

Finally, a 2017 review by Coperchini et al. of the PFOA-thyroid epidemiology literature concluded by stating that the "investigations aimed at evaluating the effects of PFOS and PFOA exposure during pregnancy on the newborn thyroid function yielded heterogeneous results, preventing univocal conclusions on which, and in what sense their thyroid function is modified." (page 116, right column). With respect to a relationship between PFOA exposure and thyroid cancer, the same authors concluded that "there was no consistent finding across all or even most studies…." (page 119, left column). In sum, there is no clear-cut reliable scientific evidence supporting a causative role of PFOA in thyroid cancer. These conclusions are directly from one of Dr. Ducatman's references. Yet Dr. Ducatman does not share the conclusions of the authors with his readers.

Liver

In the liver section of his merits report, Dr. Ducatman repeatedly fails to address the limitations of the studies that he cites.

For example, in Gallo et al. (2012), the authors noted that the principal limitations of their study was its cross-sectional methodology, making any causal inference untenable (page 6, left column). They also noted that since only a small proportion of values were outside of the normal range, it was difficult to estimate human risks (page 6, left column). More specifically, they were not confident that the observed small increase in ALT levels could lead to clinically diagnosable conditions over time (page 6, left column).

In Sakr et al. (2007), the authors noted that the cross-sectional nature of their study precluded it from being able to be used for assessing causal relationships (page 1094, right column). Furthermore, the positive associations for ALT and AST did not achieve statistical significance.

In Darrow et al. (2016), the authors concluded that their study provided a modest positive relationship between PFOA and ALT levels but "little evidence that PFOA exposure increases the risk of liver disease." (page 1232).

In Lin et al. (2010), the authors stated that their cross-sectional study design precluded them from deriving a causal inference (page 1361, right column). They also failed to indicate information on other possible chemical exposures or other factors (e.g., certain virus infections, excessive ethanol ingestion) that may affect ALT/AST values. As in the other studies cited the potential biological significance between PFOA and liver enzyme activities was considered small and subclinical.

In Gleason et al. (2015), the authors stated that the cross-sectional design "limits our ability to assess causality." (page 13, left column). In their results section the authors stated "there is no evidence of an association with PFOS and the clinical liver biomarkers ALT, AST and ALP." (page 10, right column). PFOA was positively associated with ALT/AST. However, the values were only modestly elevated.

In Yamaguchi et al. (2013), these authors also reported a modest increase in ALT/AST in relationship to PFOA levels. However, since this was a cross-sectional study it was not possible to make causal inference (page 185, right column; page 192, left column).

A study by Costa et al. (2009) representing 30 years of medical surveillance in PFOA exposed workers reported no statistical treatment effect. A 2018 clinical trial study by Convertino et al. (2018) administered doses of PFOA such that it was 10,000-fold greater than background exposure. No effects on serum ALT/AST were observed.

In his report, Dr. Ducatman claims that the above instances of modest increases in serum ALT/AST levels have population and individual significance and present a risk for non-alcoholic fatty liver disease (NAFLD) (pages 8-11, merits report). Yet he fails to note the consistent set of limitations reported by the research teams.

Simply put, the studies cited in Dr. Ducatman's reports are dominated by research methods that cannot be used to assess causal relationships. Even in these instances, there was no support that the observed very modest increase in liver biomarkers (e.g., ALT) was related to liver disease. Dr. Ducatman provides no scientific methodology by which he transformed the above non-significant responses into induced liver disease that would be more likely than not to occur.

Dr. Ducatman also cites several animal studies with exposure to PFOA to support his asserted risk of developing hepatic steatosis. Yet, unremarked by Dr. Ducatman, an examination of these papers reveals that they were short-term high dose experiments. The exposures were about 3-4 orders of magnitude greater than typical human exposures. These studies are not relevant to humans. No consideration was given to the capacity of the different strains of mice or rats to reliably extrapolate to humans. Likewise, no consideration was given in the assessment to the nature of the total dose and dose-rate.

In the case of Yang et al. (2014), the entire daily dose was administered in one gavage setting. Further, no consideration was given to the possibility that handling the model by the technician could affect susceptibility to agent induced liver damage (Calabrese, 2001). While these studies appear to be in the realm of "proof of concept" hypothesis development, they are not related to the issue of establishing risk in the low dose zone, at issue here.

Hyperlipidemias

Dr. Ducatman states that PFOA exposure has been repeatedly associated with changes in lipid metabolism, noting higher total cholesterol and LDL cholesterol in multiple studies for adult men/women, pregnant women and children. He further states that such blood lipid changes have substantial population and individual significance and that more people living in these communities will eventually need treatment for high cholesterol. These nearly 20 cited human studies were then complemented with about a dozen animal studies assessing the effects of PFOA on lipid metabolism and related disease pathology with focus on steatosis, that is, the abnormal retention of lipid within a cell, typically due to an impairment of the normal process of synthesis and elimination of triglyceride fat.

Nonetheless, Dr. Ducatman's reports fail to address the issue of epidemiological study design and the capacity (or lack thereof) to make causal inferences. A substantial number of the cited studies (Fu et al., 2014; Frisbee et al., 2010; Eriksen et al., 2013; Matilla-Santander et al., 2017; Steenland et al., 2009; Sakr et al., 2007; Zeng et al., 2015) employed cross-sectional designs. The authors recognized the key limitations of this approach, which preclude deriving causal inferences. For example, Fu et al. (2014) stated "it was impossible to establish a causal inference due to the cross-sectional nature of this study." (page 251, left column). Likewise, Sakr et al. (2007) stated that cross-sectional studies "can not be used for determining causality." (page 1094, right column).

In addition to the widespread use of cross-sectional studies that preclude a causality argument, Dr. Ducatman fails to address the issue of variability in serum cholesterol estimates, including their magnitude and underlying causes and their distribution within populations. The published literature indicates that there can be large differences in serum cholesterol levels within the same subject with repeat measurement over consecutive days (Cooper et al., 1992; Craig et al., 2000; Hegsted and Nicolosi, 1987; Nigam, 2011).

If one assumes a mean serum cholesterol level of 220 mg/dl and a mean intra-individual standard deviation (SD) of only 5% of the mean, then one would expect a single measurement to occur within 2 SDs above or below the true mean. More specifically, a single sample (as reported in essentially all studies cited by Dr. Ducatman on this matter) with a mean of 220 mg/dl would be expected to fall between 200 and 240 mg/dl. Even this broad range estimate is likely to be very optimistic because the SD of 5% is only about twice the analytic error in highly experienced laboratories and many people will show greater variation than 5%.

The reported studies cited by Dr. Ducatman did not standardize for the time of day the sample was tested, fasting behavior, activities prior to blood drawing, information on medicinal drug intake, and numerous other potential modifying factors. Thus, one cannot reliably evaluate possible positive or negative bias in these cited studies and how these factors may have affected the reported findings. The published articles generally did not provide information on subject diets, obesity, smoking, exercise, alcohol intake, blood pressure and other factors that may impact serum cholesterol, and how these factors may have affected the reported associations. Due to these limitations and others, one cannot reliably draw any causal inference from such studies. Yet Dr. Ducatman does not address these limitations in his reports.

Uric Acid

In his class certification and merits reports, Dr. Ducatman cites multiple studies for the proposition that PFOA exposure is associated with increased serum uric acid levels. In his merits report, he claims that the elevated levels had significance at the population and individual levels. He also states that people of all ages would experience increased serum uric acid levels due to the PFOA exposure. This led Dr. Ducatman to speculate on possible enhanced medical risks such as gout and kidney disease.

Based on this argument, he recommends uric acid testing in the proposed medical monitoring program. Yet most of the cited epidemiological studies acknowledged their cross-sectional methodological nature. For instance, as Qin et al. (2016) noted, their "findings cannot establish a causal relationship between PFASs and serum uric acid levels because of the nature of the cross-sectional study design." (page 523, left column). Similar comments were offered by Geiger et al., 2013, (page 1259, right column).

The papers cited by Dr. Ducatman noted a possible uric acid increase of about 0.2 to 0.4 mg/dl. Several reports indicated that there is considerable variation in daily and monthly serum uric acid values with an approximately 10% standard deviation (Yu et al., 2004). In addition to day-to-day variation, there is also considerable diurnal variation with highest values reported for morning, decreasing by about 20% at mid-afternoon (Devgun and Dhillon, 1992).

None of the papers cited by Dr. Ducatman addresses the issue of time of the day that individuals had blood drawn for uric acid value determination. None of the cited papers obtained information on weight, exercise, diet, fasting, and season, factors that may impact uric acid measurement values. The failure of the reported studies to address key variables that affect serum uric acid variation creates a significant issue when one is attempting to evaluate very small reported changes. Yet Dr. Ducatman does not address these issues in his reports.

Notably, Beavers et al. (2014) indicated that a moderate exercise program for 12 months in an older population induced an increase of serum uric acid similar to that claimed to the PFOA. The increase in the exercise study was of a magnitude such that it would not be considered clinically meaningful (page 7, first full paragraph). However, the modest increase in uric acid was associated with improved muscle function and better physical performance scores in older subjects (page 7, last sentence, first full paragraph).

Further Discussion of Methodological Issues

The opinions of Dr. Ducatman are at such a general and superficial level as to preclude an evaluation that would permit the capacity to address questions of possible causality and, in turn, whether a medical monitoring program could be justifiable. His reports provide no standard for causality evaluation to be followed or even guided; no methodology to be applied, even with

possible modifications; no description of the studies, including their design, methodology, strengths or limitations; and no assessment of the data adequacy/quality and data evaluation methods. On page 10 of his deposition Dr. Ducatman replied, "Yes," when asked if he believed that "scientists should describe their methods and explain their reasoning so that others can understand how the data were analyzed and how the conclusions were reached." Yet in his written opinions for this case he failed to follow this principle, making it impossible to evaluate the basis of his opinions.

Dr. Ducatman does not provide any description or assessment of the statistical analyses and information on statistical significance and their interpretation for application and how it relates to his opinions. His reports do not provide an integrative assessment of the data that would permit an evaluation of multiple and diverse epidemiological studies for use in deriving opinions. (See Ducatman deposition, pages 82-83.)

Dr. Ducatman's reports fail to evaluate the occurrence of other peroxisome proliferator receptor activating agents that humans are exposed to in their diets or in widely used medicines that act via receptor-based mechanisms (peroxisome proliferator α and γ receptors). He does not address the likelihood of interactions between peroxisome proliferator activating agents with other chemicals and drugs, such as the components in wine, marijuana, and other widely used substances, and how such exposures may affect study outcomes, interpretations, and uncertainty of his opinions.

The human dietary sources of PPARγ ligands from plants are vast and substantial. These include commonly ingested products, including pomegranate, apples, clove, cinnamon, thyme, green coffee, bilberry, chili pepper, nutmeg, cacao, caraway, licorice, sage, rosemary, various

mushrooms, curry, ginseng, black pepper, red onions, dill, white cabbage, sauerkraut juice, peas, lavender, and many other plant materials (Mueller and Jungbauer, 2009) (page 660, abstract).

A common PPARγ drug used to treat type 2 diabetes is rosiglitazone, with an average daily dose of 4-8 mg. Approximately 100 milliliters (i.e., a little more than 3 liquid ounces) of many red wines contain the equivalent of 1.8 to 18 mg of the rosiglitazone, corresponding to 25-400% the daily dose of this drug. Since a typical glass of wine contains about 6 ounces, this dose of such PPARγ receptor activators alone can be substantial (Zoechling et al., 2011). In fact, the anti-diabetes effects of grape seed extracts and the cardioprotective effects of red wine have been widely reported in the biomedical and medical literature (El-Alfy et al., 2005; Karthikeyan et al., 2007; Pinent et al., 2006). Likewise, the effects of PPARγ on macrophages (Akbiyik et al., 2004) accounts for some of its ability to suppress inflammation and reduce the occurrence of inflammatory diseases. The PPARγ ligands are now widely known to facilitate the occurrence of macrophage polarization, reprogramming macrophages toward anti-inflammatory phenotypes that can be protective in all organs. These findings have important public health and medical implications since so many diseases and aging processes have an inflammatory component (Jungbauer and Medjakovic, 2012; Wang et al., 2014; Zhao et al., 2016).

There is also widespread and substantial exposure to natural products for targeting and activating PPARα receptors. Such activation is seen with caraway seeds, chili pepper, nutmeg, licorice, black and white pepper, paprika, coriander, saffron, various teas, and other products. It is now widely recognized that diets rich in fruits and herbs and spices provide substantial exposures to PPARα agonists, which can have numerous biological effects that might affect one's lipid profile and inflammation status (Mueller et al., 2011; Rigano et al., 2017; Jungbauer and Medjakovic; Fidaleo et al., 2014).

Nonetheless, Dr. Ducatman's reports fail to address the occurrence of dietary peroxisome proliferator agonists and how they may affect biological processes and their biomedical and therapeutic applications. His reports do not assess how such exposures occur and differences amongst individuals and study populations. Consequently, these omissions represent a gaping hole in his interpretation of published PFOA studies.

In fact, none of the epidemiological studies of PFOA cited by Dr. Ducatman addresses the issue of dietary exposures to peroxisome proliferators, how they may distribute in the population, and how they may change over time in lives of the studied subjects. The failure of the epidemiologic studies on PFOA to collect data on dietary exposures to these agents is an important limitation in such studies. Likewise, the lack of any consideration of this issue by Dr. Ducatman reveals that his opinions have not considered alternative causal interpretations for study variation and individual variation that relate to this central causality issue.

Dr. Ducatman's reports do not address the occurrence of genetic polymorphisms relating to peroxisome proliferating agents and how these may vary within human populations and affect study outcomes and impact any proposed medical monitoring program (Contreras et al., 2013). These issues speak to the fact that Dr. Ducatman's reports not only fail to assess properly the studies on PFOA that were available, but also fail to consider other relevant scientific questions such as alternative causality.

CONCLUSION

The opinions of Dr. Ducatman concerning the relationship between PFOA and toxic effects in humans lack scientific merit and cannot be accepted. Likewise, there is no scientific basis to support his recommendation for a medical monitoring program.

I reserve the right to use graphics or other exhibits to further address the matters discussed herein and to supplement this report based on new or additional data.

COMPENSATION AND TESTIMONIAL HISTORY

I am being compensated at the rate of $600/hour for all activities associated with this matter. To the best of my recollection, during the previous four years, I have testified as an expert at trial or by deposition in the following cases: Abernathy et al. v. Occidental et al. (2017) – Deposition; Forcellati v. Hyland's (2015) - Court Testimony – Los Angeles, CA; Johnson et al. v. Motorola Inc. (2015) – Deposition.

DECLARATION

This report contains a complete statement of all opinions I will express and the basis and reasons for them, as well as the facts or data I considered in forming these opinions. I declare under penalty of perjury under the laws of the United States that the foregoing is true and correct to the best of my knowledge.

Dated: May 7, 2018

Edward J. Calabrese, Ph.D.

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EXHIBIT A

EDWARD J. CALABRESE, PH.D.

CURRICULUM VITAE

March 2018

I. SUMMARY:

- Professor of Toxicology at the University of Massachusetts, Amherst since 1976.
- Board Certified in general toxicology by the Academy of Toxicological Sciences since 1982.
- Over 825 publications in peer-reviewed journals.
- Among the most highly cited papers in the entire history of several leading toxicology journals.
- Over 800 invited presentations at major conferences and University seminars.
- Author or Co-Author of 26 books.
- Editor or Co-Editor of over 40 monographs and/or conference proceedings.
- Consultant to most environmentally oriented federal agencies.
- Member of multiple national research council expert committees such as the Safe Drinking Water Committee, Air Cabin Safety Committee, and Food and Nutrition Committee.
- Consultant to numerous major U.S. corporations and trade associations.
- Extramural funding since 1976 from all sources exceeds 30 million dollars.
- Founding Editor-in-Chief Human and Ecological Risk Assessment.
- Founding Editor-in-Chief Dose-Response Journal.
- Recipient of the Springer Award for the body of work on hormesis, 2010.
- Honorary Doctor of Science Degree, McMaster University 2013.
- Awarded the Petr Beckmann Award from Doctors for Disaster Preparedness 2014.
- Advisory Board for the first graduate training program focused on hormetic mechanisms, Friedrich-Schiller-University, Jena, Germany 2011 to present.

Table 1. Number of Publications Per Year

				2018 = 19	2017 = 32
2016 = 13	2015 = 20	2014 = 22	2013 = 22	2012 = 19	2011 = 10
2010 = 27	2009 = 13	2008 = 28	2007 = 9	2006 = 13	2005 = 15
2004 = 10	2003 = 20	2002 = 15	2001 = 42	2000 = 22	1999 = 15
1998 = 22	1997 = 14	1996 = 17	1995 = 18	1994 = 21	1993 = 25
1992 = 17	1991 = 20	1990 = 25	1989 = 25	1988 = 26	1987 = 20
1986 = 29	1985 = 32	1984 = 12	1983 = 22	1982 = 20	1981 = 11
1980 = 23	1979 = 24	1978 = 13	1977 = 11	1976 = 5	1975 = 2
1974 = 10	1973 = 1	1972 = 1	1968 = 1		Total = 825

II. BIOGRAPHICAL SKETCH:

Edward J. Calabrese is a Professor of Toxicology at the University of Massachusetts, School of Public Health and Health Sciences, Amherst. Dr. Calabrese has researched extensively in the area of host factors affecting susceptibility to pollutants, and is the author of over 800 papers in scholarly journals, as well as more than 10 books, including Principles of Animal Extrapolation; Nutrition and Environmental Health, Vols. I and II; Ecogenetics; Multiple Chemical Interaction; Air Toxics and Risk Assessment; and Biological Effects of Low Level Exposures to Chemical and Radiation. Along with Mark Mattson (NIH) he is a co-editor of the recently published book entitled Hormesis: A Revolution in Biology, Toxicology and Medicine. He has been a member of the U.S. National Academy of Sciences and NATO Countries Safe Drinking Water committees, and on the Board of Scientific Counselors for the Agency for Toxic Substances and Disease Registry (ATSDR). Dr. Calabrese also serves as Chairman of the Biological Effects of Low Level Exposures (BELLE) and as Director of the Northeast Regional Environmental Public Health Center at the University of Massachusetts. Dr. Calabrese was awarded the 2009 Marie Curie Prize for his body of work on hormesis. He was the recipient of the International Society for Cell Communication and Signaling-Springer award for 2010. He was awarded an Honorary Doctor of Science Degree from McMaster University in 2013. In 2014 he was awarded the Petr Beckmann Award from Doctors for Disaster Preparedness.

Over the past 20 years Professor Calabrese has redirected his research to understanding the nature of the dose response in the low dose zone and underlying adaptive explanatory mechanisms. Of particular note is that this research has led to important discoveries which indicate that the most fundamental dose response in toxicology and pharmacology is the hormetic-biphasic dose response relationship. These observations are leading to a major transformation in improving drug discovery, development, and in the efficiency of the clinical trial, as well as the scientific foundations for risk assessment and environmental regulation for radiation and chemicals.

CURRICULUM VITAE

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III. ACADEMIC TRAINING		
University of Massachusetts, Amherst, MA	1972-1974 – Education Science Ed.	Ed.D. 1974
University of Massachusetts, Amherst, MA	1971-1973 – Physiology/Toxicology, Entomology Department	Ph.D. 1973
State College of Bridgewater, Bridgewater, MA	1969-1971 – Biology	MA 1972
State College of Bridgewater, Bridgewater, MA	1964-1968 - Biology	BA 1968

IV. WORK EXPERIENCE

Graduate Program Director, Environmental Health Sciences Department, December 2003-2004.

Division Chair, Environmental Health Sciences Division, December 2003-2006.

Director - Northeast Regional Environmental Public Health Center, October 1985-Present.

Professor - Promoted from Associate Professor, June 1982-Present.

Associate Professor - Promoted from Assistant Professor, June 1980.

Assistant Professor - September 1976 - Environmental Health Sciences Program, Division of Public Health, University of Massachusetts, Amherst, MA. Duties include: teaching introductory and advanced courses in environmental toxicology, directing thesis research.

Assistant Professor - July 1974-August 1976 - Department of Occupational and Environmental Medicine, University of Illinois, School of Public Health, and Assistant Director of the Environmental Health Resource Center. Duties included: the identification and quantification of present and potential environmental health hazards within the state, the development and review of environmental health legislation, standards and regulations, testimony at regulatory and legislative hearings on standards of environmental quality and teaching courses in environmental health.

Environmental Research Director for the Massachusetts Public Interest Research Group - December 1973-June 1974. Duties included: determination of research and educational goals of the organization, direction of student research projects, direction of Water Quality Training Institutes throughout Massachusetts.

Adjunct Professor - Southwest Residence College - University of Massachusetts. January 1974. Taught environmental science courses to undergraduate and graduate students.

Assistant Professor - Fall 1973 - North Adams State College, North Adams, MA. Biology Department - taught Ecology, Evolution, and Introductory Biology.

V. GRANTS AND RESEARCH FUNDING

Principal Investigator. Coca-Cola Company. Environmental Health Sciences 2-22-2016 – present ($25,000).

Principal Investigator. Air Force Office of Scientific Research. Enhancing Biological Performance: Occurrence, Mechanisms and Applications. 2013-2018. ($1,197,558).

Principal Investigator. Exxon Mobil Foundation. Research and Education work on the topic area of hormesis. 2014 ($125,000).

Principal Investigator. Samueli Institute. Conference on Dose-Response. 2013-2014 ($15,000).

Principal Investigator. ExxonMobil. Hormesis Research. 2007-2013. ($150,000 per year).

Director. Hormesis Conference general support. Multiple public and private organizations. 2010-2013. (Approximately $50,000).

Principal Investigator. Air Force Office of Scientific Research. Conference on Adaptive Responses and their Biomedical Applications. 2012. ($25,544).

Principal Investigator. Air Force Office of Scientific Research. Conference on Adaptive Responses and their Biomedical Applications. 2011. ($25,580).

Principal Investigator. Lounsbery Foundation. Development of an Integrative Mechanistic Framework. 2010-2012. ($25,000)

Principal Investigator. Air Force Office of Scientific Research. Chemical/Radiation Hormesis Database, Evaluation of Hormetic Mechanisms & Their Biomedical and Risk Assessment Implications. 2008-2010. ($299,371).

Director. Hormesis Conference general support. Multiple public and private organizations. 2008-2009. (Approximately $120,000).

Principal Investigator. Air Force Office of Scientific Research. Chemical/Radiation Hormesis Database, Evaluation of Hormetic Mechanisms & Their Biomedical and Risk Assessment Implications. 2007. ($84,778).

Principal Investigator. Air Force Office of Scientific Research. Chemical/Radiation Hormesis Database, Evaluation of Hormetic Mechanisms & Their Biomedical and Risk Assessment Implications. 2007. ($199,845).

Director. Hormesis Conference general support. Multiple public and private organizations. 2007. (Approximately $150,000).

Director. Hormesis Conference general support. Multiple public and private organizations. 2006. (Approximately $100,000).

Principal Investigator. Alfred P. Sloan Foundation. Hormesis Center. 2004-2007. ($45,000).

Principal Investigator. Dow Chemical Co. Distributions for Monte-Carlo Soil Ingestion Risk Assessment. 2004-2007. ($160,470).

Principal Investigator. Lounsbery Foundation. Workshop to Create a Hormesis Institute/Center. 2005-2007. ($75,000).

Principal Investigator. ExxonMobil. Hormesis Research. 2006. ($150,000).

Principal Investigator. Air Force Office of Scientific Research. Chemical/Radiation Hormesis Database, Evaluation of Hormetic Mechanisms & Their Biomedical and Risk Assessment Implications. 2006. ($214,645).

Principal Investigator. ExxonMobil. BELLE – Chemical Hormesis Database. 2005. ($150,000).

Principal Investigator. Air Force Office of Scientific Research. Chemical/Radiation Hormesis Database, Evaluation of Hormetic Mechanisms & Their Biomedical and Risk Assessment Implications. 2005. ($211,026).

Principal Investigator. U.S. Department of Energy. International Conference – Hormesis Implications for Toxicology, Medicine, and Risk Assessment. 2005-2006. ($5,000).

Principal Investigator. Dow Chemical Co. Distributions for Monte-Carlo Soil Ingestion Risk Assessment. 2004-2006. ($160,470).

Principal Investigator. Alfred P. Sloan Foundation. Hormesis Center. 2004-2006. ($45,000).

Principal Investigator. U.S. Department of Energy. Non-Linear Dose Response Relationship in Biology, Toxicology and Medicine. 2004-2005. ($20,000).

Principal Investigator. General Electric Foundation. BELLE Initiative. 2004. ($100,000).

Principal Investigator. ExxonMobil. BELLE – Chemical Hormesis Database. 2004. ($75,000).

Principal Investigator. Air Force Office of Scientific Research. Chemical/Radiation Hormesis Database, Evaluation of Hormetic Mechanisms & Their Biomedical and Risk Assessment Implications. 2004. ($174,302).

Principal Investigator. U.S. Department of Energy. Non-Linear Dose Response Relationship in Biology, Toxicology and Medicine. 2003-2004. ($12,500).

Principal Investigator. Florida Power and Light. Assessment of Arsenic Bioavailability in Humans. 2002-2003. (~$110,000).

Principal Investigator. Air Force. Toxicological Assessment of Hormesis. 2001-2003. ($450,000).

Principal Investigator. US EPA/American Chemical Council. Soil Ingestion in Construction Workers. 2001-2003. ($750,000).

Co-Principal Investigator. Health Risks and Fish Consumption from the Pasiac River. 2001-2002. ($125,000).

Principal Investigator. CA EPA. Single Exposure Carcinogen Database Update and Evaluation. 2002. ($50,000).

Co-Director. 11th Annual Soil and Groundwater Conference. San Diego, CA. March 2002. ($100,000).

Co-Director. 18th Annual Soil, Groundwater and Sediment Contamination Conference. University of Massachusetts. October 2001. ($125,000).

Principal Investigator. Conference on Non-Linear Dose-Response. Multiple sponsors (EPA, NIEHS, AWWARF, Air Force, and other). June 2001. ($150,000).

Co-Director. International Conference on Contaminated Soil, Sediment, and Groundwater. London. August 2000. ($300,000).

Co-Principal Investigator. Soil ingestion workshop/assessment. U.S. EPA. June/July 2000. ($50,000).

Principal Investigator. Soil ingestion in construction workers. U.S. EPA/CMA. October, 1999 ($650,000).

Principal Investigator. Development of an ionizing radiation hormesis database. Nuclear Regulatory Commission. September 1997 - September 1999 ($188,000).

Principal Investigator. Biological effects of low level exposures. Three year cooperative agreement. Reviewed once, 1999. Nuclear Regulatory Commission, 1996-1998, 1999-2001. ($60,000 or $20,000/year).

Principal Investigator. Assessment of soil ingestion in children. Health Canada. January 1999 ($6,500).

Principal Investigator. Biological effects of low level exposures (BELLE). From multiple sponsors. 1997, 1998, 1999, 2000, 2001, 2002, 2003, 2004. (approx. $120,000/year from multiple sources).

Co-Principal Investigator. Florida Power and Light. Biological effects of arsenic contaminated soil. January 1998 ($100,000), March 1999 ($50,000).

Principal Investigator. ARCO. Assessment of the role of particle size on soil ingestion estimates in children. June 1997 ($150,000).

Principal Investigator. Health Research Foundation (Japan). Biological effects of low level exposures. September 1997 ($15,000).

Principal Investigator. U.S. Air Force. Assessment of the societal and scientific implications of hormesis. October 1997 - October 2000 ($345,000).

Principal Investigator. U.S. EPA. Single exposure carcinogen database. October 1997 – May 1999 ($75,000).

Principal Investigator. GE Foundation. Biological effects of low level exposures (BELLE). October 1997 ($15,000).

Co-Principal Investigator. EPA. Assessment of groundwater contamination by MTBE. September 1997 ($43,000).

Principal Investigator. Exxon. Biological effects of low level exposures. 1996-1999 $20,000/year. ($80,000).

Principal Investigator. Dow-Corning. Biological effects of low level exposures. 1996-1999 $10,000/year. ($40,000).

Principal Investigator. Canadian Electric Utilities. Biological effects of low level exposures. 1996 ($10,000).

Co-Director. Bitor-Venezuela. Evaluation of the endocrine disruption potential of surfactants. June 1996 ($447,000).

Co-Principal Investigator. Massachusetts Department of Environmental Protection. Determination of heavy metal background levels. June 1996 ($23,000).

Principal Investigator. ARCO. Assessment of the role of particle size on soil ingestion estimates in children. June 1996 ($150,000).

Principal Investigator. Radiation, Science and Health, Inc. Critical assessment of selected literature on radiation hormesis. December 1996 ($26,000).

Principal Investigator. Environmental effects of Orimulsion. December 1996 ($836,000).

Principal Investigator to support BELLE related activities. January 1995. RJReynolds, Inc., $25,000; Electric Power Research Institute, $10,000; Dow Corning, $10,000; and Canadian Electric Utilities, $10,000.

Principal Investigator. RJReynolds, Inc. The effects of low levels of chemical agents on biological responses. February 1995 ($25,000).

Principal Investigator to assess soil ingestion in children living in Northwest of the U.S. ARCO. September 1992 - June, 1996 ($748,000).

Principal Investigator. Louisiana DEQ. Assessment of soil ingestion in children. June 1995 ($50,000).

Principal Investigator. US EPA. An evaluation of gender differences in susceptibility to toxic substances. June 1995 ($55,000).

Principal Investigator. US EPA. Single exposure carcinogen database. October 1995 ($75,000).

Principal Investigator. Health Canada. Develop new methodologies to assess human high risks. November 1994 ($60,000).

Principal Investigator to direct BELLE activities. EPRI, Dow Corning, Center for Indoor Research, and EPA. October 1994 ($55,000).

Principal Investigator. Florida Power and Light. Development of a framework to conduct an ecological risk assessment on Tampa Bay. April 1994 ($140,000).

Principal Investigator. Gillette, Inc. Support of BELLE-related activities. May 1994 ($3,000).

Principal Investigator. Florida Power and Light. Assess the effects of several types of fuel oil on red blood cells. September 1994 ($31,000).

Co-Director of a series of conferences on petroleum contaminated soil. Held at the University of Massachusetts, Amherst. 1985, 1987, 1988, 1989, 1990, 1991, 1992, 1993, 1994, 1995, 1996, 1997, 1998, 1999, 2000, 2001, 2002. Approximately $100,000/conference from external co-sponsors.

Co-Director of a series of conferences on soil and groundwater contamination. Held in the greater Los Angeles area. 1989-2002. $100,000/year.

Principal Investigator on a grant to assess interspecies differences in hepatic peroxisomes proliferation and its role in the development of fish tumors. Department of Defense, U.S.A. April 1988-1993 ($749,000).

Florida Power and Light. Critical Evaluation of the PM_{10} standard. November 1993 ($20,000).

Principal Investigator to direct BELLE activities: EPRI, Dow Corning, Center for Indoor Research, and others. April 1993 (approx. $50,000).

Principal Investigator to assess single exposure carcinogens. ATSDR/September 1993 ($50,000).

Principal Investigator to assess the prevalence of soil pica in children and soil ingestion in children with soil pica. State of Colorado. July 1992 ($151,000).

Principal Investigator to direct the development of a newsletter on the Biological Effects of Low Level Exposures (BELLE). U.S. EPA. September 1992 ($60,000).

Director of the Council for Health and Environmental Safety of Soils Funded by EPA, ATSDR and other organizations. 1988 – 1992 ($150,000/yr.)

Principal Investigator. U.S. EPA. Lead Training Center. March 1992 ($320,000); October 1993 ($220,000); October 1994 ($290,000).

Co-Director of National Conference on Hydrocarbon Contaminated Soils. From multiple agencies/organizations. ($70,000).

Co-principal Investigator - Development of risk assessment methods for human and ecological risks. Health and Welfare Canada. April 1 1992 ($75,000).

Co-principal Investigator for Regional Lead Training Center. U.S. EPA. April 1992 ($250,000).

Principal Investigator to conduct national conference on the Biological Effects of Low Level Exposures to Chemicals and Radiation. NIEHS. April 1992 ($10,000).

Principal Investigator to support research activities concerning the biological effects of low level exposures (BELLE). Ontario Hydro. January-May 1992 ($20,000); RJR-Nabisco ($35,000); EPRI ($10,000).

Principal Investigator to assess the effects of selected oxidant stressor contaminants on red blood cells. State of Colorado. May 1992 ($44,000).

Principal Investigator to assess factors assessing the siting of waste sites in the U.S. Waste Management Inc. June 1992 ($200,000).

Principal Investigator to assess environmental factors affecting stream health. Wyman-Gordon, Co. July 1992 ($135,000).

Co-Director of the Hydrocarbon Contaminated Soil and Groundwater Conference. Newport Beach, California. 1991 - co-sponsorship $100,000 (approx.).

Principal Investigator to unrestricted support on predictive toxicology. Proctor and Gamble. June 1991 ($5,000).

Co-principal Investigator to develop a toxicological based risk communication program for lead in water. U.S. EPA. August 1991 ($50,000).

Co-Director of the 6th Annual Hydrocarbon Conference. Sept. 1991 (combined sponsorship $100,000. From multiple agencies, federal, state and private sector).

Principal Investigator of a project to differentiate soil and dust ingestion in children. U.S. EPA. Sept., 1991 ($50,000).

Principal Investigator to support research activities concerning the biological effects of low level exposures (BELLE). Dow Chemical. November 1991 ($5,000).

Principal Investigator to support research activities concerning the biological effects of low level exposures. RJR Nabisco, Inc. July 1990 ($45,000).

Principal Investigator-Evaluation of the health basis for EPA's regulations of SOTs and IOCs in drinking water. American Water Works Association Research Foundation. July 1990 ($100,000).

Principal Investigator on contract to assess the relative potency of methemoglobin forming agents. EPA. July 1990 ($28,000).

Principal Investigator-Methemoglobin forming agents: Toxicologic and risk assessment. EPA. August 1990 ($28,000).

Principal Investigator to support research activities concerning the biological effects of low level exposures. Dow Chemical. November 1990 ($10,000).

Principal Investigator to support research activities concerning the biological effects of low level exposures. The Electric Power Research Institute. December 1990 ($10,000).

Co-Director of the Hydrocarbon Contaminated Soil and Groundwater Conference. Newport Beach, California. 1990 - co-sponsorship $100,000 (approx.).

Principal Investigator of a contract to assess the Public Health risks associated with medical waste. Funded by the Rockefeller Institute of Government, Albany, New York. January 1989 ($15,000).

Co-Principal Investigator on a grant to assess factors affecting heavy metal tissue distribution in selected fish species. General Electric. July 1989 ($112,500).

Co-Principal Investigator on a grant to assess public health aspects of soil contaminated with petroleum. U.S. EPA. July 1989 ($43,000).

Principal Investigator to continue research on how to estimate how much soil children ingest. Gradient Corporation. August 1989 ($35,000).

Director of a conference on drinking water and health. American Water Works Association Research Foundation. September 1989 ($10,000).

Principal Investigator of a contract to assess the methodological approaches for establishing an Air Toxic Programs. Rohm and Haas, Inc. Part 1 - January 1987 ($60,0000. Part 2 - January 1988 ($60,000).

Principal Investigator on a grant to develop an approach for assessing human risk for soil contamination. Hercules Corporation. January 1988 ($10,000).

Principal Investigator of a contract to assess environmental exposure from the application of lawn care chemical treatment practices. Massachusetts Department of Food and Agriculture. January 1987 - June 1987 $75,000; July 1987 - June 1988 ($75,000).

Director on a grant from Proctor and Gamble in the general area of research in animal extrapolation. July 1988 ($5,000).

Principal Investigator of a grant to assess the amount of soil children consume. Syntex, Corporation. August 1988 ($25,000).

Principal Investigator of a study to assess the environmental and public health effects of soils contaminated with petroleum products including disposal options. Mass. Depart. of Environ. Engineering. July 1986 - June 1987 ($108,000).

Director of workshop on risk assessment for aerial spraying of insecticides for control of gypsy moths. U.S.D.A. - Forest Service. January 1986 ($12,000).

Co-principal Investigator of a grant to assess the effects of acid rain on selected freshwater fish species. Massachusetts Fish & Wildlife Service. May 1986 ($7,000).

Co-principal Investigator of a contract to assess the environmental and public health implications of disposal options for petroleum contaminated soil. Edison Electric Institute. July 1986 ($50,000).

Co-principal Investigator to establish an aquatic toxicology research program in the School of Public Health. Funded by the Mass. Department of Fisheries and Wildlife. July 1986 ($100,000/year).

Principal Investigator of a study to assess the environmental and public health effects of soils contaminated with petroleum products including disposal options. Mass. Depart. of Environ. Engineering. September 1984 - June 1985 ($71,000). July 1985 - June 1986 ($76,000).

Director on a grant from Proctor and Gamble in the general area of research in animal extrapolation. August 1986 ($5,000), an additional $5,000.00 was received in July 1987.

Principal Investigator of a grant to assess the amount of soil children consume. Syntex, Corporation. August 1986 ($344,000).

Co-principal Investigator of the 3-year grant to assess the aquatic toxicity of chlorination of waste water treatment plants. Mass. Water Pollution Control Assoc. September 1986 ($90,000).

Director of EPA sponsored conference on the Environmental and Health effects of Ozone. U.S. EPA. October 1986 ($10,000).

Principal Investigator of a grant from the University of Illinois - Effects of ozone on mice with low levels of glucose-6-phosphate dehydrogenase in red cells. January 1985 ($5,000).

Principal Investigator of a study entitled "The Effect of Environmental pH and Modifying Factors on the Reproduction of Rainbow Smelt." Massachusetts Fish and Wildlife Service. January 1985 ($9,873).

Director of a contract to provide toxicological and risk assessment consultation and research to the Connecticut State Health Department. February 1985 ($90,000).

Principal Investigator of a study to assess possible reproductive hazards in the semi-conductor industry. Digital Corporation: Phase 1 - July 1984 ($244,000); Phase 2 - March 1, 1985 ($194,000).

Director of the Northeast Regional Environmental Health Center, sponsored by the six New England States. Starting October 1985 (goal of $250,000/year).

Principal Investigator on the assessment of the occurrence of biological factors affecting interindividual variation in response to toxic substances. Hercules Corporation. October 1985 ($11,000).

Director of a national conference on "Environmental and Public Health Effects of Soils Contaminated with Petroleum Products." Funded by the Massachusetts Department of Environmental Quality Engineering, EPRI, ARCO, Northeast Utilities and other companies. October 1985 ($50,000).

Director of a contract to assess the public health hazards associated with leaking underground storage tanks. EPRI. October 1985 ($20,000).

Co-Investigator of a study to assess the possibility of using surrogate parameters in monitoring for the presence of volatile organic contaminants in drinking water. American Water Works Association Research Foundation. October 1984 ($60,000).

Principal Investigator of a study to assess the effects of elevated levels of sodium in drinking water on school children. Massachusetts Department of Environmental Quality Engineering. June 1983 ($10,000).

Developed the concept and proposal for a state-supported Environmental R & D Center. It was funded by the Massachusetts Legislature in July 1983 for up to $500,000 per year.

Director of a grant from the U.S. EPA to conduct an International Conference on Cardiovascular Disease and Inorganic Constituents in Drinking Water. August 1983 ($65,000).

Director of a contract from the Massachusetts Department of Environmental Quality Engineering to assess the impact of several plastics manufacturing plants on ambient air quality. September 1982 ($5,068).

Principal Investigator of a contract to assess government policy with respect to genetic screening in the workplace. U.S. Congress' Office of Technology Assessment. January 1982 ($7,400).

Principal Investigator of a Biomedical Research Grant from the University of Massachusetts Graduate Research Council to study the development of an animal model to simulate human hereditary blood disorders (i.e., G-6-PD deficiency). April 1982 ($5,000).

Director of a quarterly newsletter entitled "Health Effects Update" for members of the American Water Works Association. May 1982 ($20,000/year).

Principal Investigator of a grant to investigate the efficacy of the guinea pig heterologous model to predict the effects of ozone on human erythrocytes with a G-6-PD deficiency. Hoffmann-LaRoche, Inc. June 1982 ($10,000).

Principal Investigator of a grant to study the effects on blood pressure of a reduction in sodium in drinking water from 120 ppm to 25 ppm. American Water Works Research Foundation. June 1982 ($29,000).

Principal Investigator on a study designed to evaluate the effect of ascorbic acid supplementation on the body burden of lead. Hoffmann-LaRoche, In. July 1982 ($14,700).

Co-principal Investigator on an unrestricted grant from the State of Massachusetts Department of Environmental Quality Engineering to study the potential of organics in drinking water as pollutants in household air. November 1981 ($600).

Principal Investigator of a grant to investigate the effects of variable dietary ascorbic acid intake on the toxicity of a proposed toxic ozone intermediate on human subjects (in vitro). Hoffmann-LaRoche, Inc., N.J. December 1981 ($10,000).

Director of a $41,000 grant from the U.S. EPA to conduct an International Conference on Cardiovascular Disease and Drinking Water during May 1979.

Principal Investigator on a contract from the U.S. EPA to provide a critical assessment of the epidemiological and toxicological studies concerning the health implications of widespread use of diesel fuel. June 1979 ($9,500).

Co-principal Investigator on a contract from the U.S. EPA to evaluate the effects of chlorite on the kidney, blood pressure, and blood parameters in adult and neonate rats and mice. December 1979 ($176,198).

Co-principal Investigator on a grant from the U.S. EPA to conduct a study on the effects of elevated levels of sodium in drinking water on cardiovascular function. March 1978 ($950,000).

Director of a $24,000 grant from the U.S. EPA to conduct an International Conference on the Effects of Pollutants on High Risk Groups during June 1978.

Principal Investigator on a grant from the U.S. EPA to conduct a study on the effects of ozone and nitrogen dioxide on mice with low levels of glucose-6-phosphate dehydrogenase in their red cells. June 1978 ($211,000).

Co-principal Investigator on a grant from the U.S. EPA to conduct a study on the effects of chloramines, chlorite, and copper on pregnant female mice with red cells having low levels of glucose-6-phosphate dehydrogenase. July 1978 ($95,000).

Co-principal Investigator on a U.S. EPA grant to evaluate the effect of chlorine dioxide disinfection on neonates born during 1946 in a community that temporarily adopted the use of chlorine dioxide for disinfection. 1978 ($50,000).

Co-principal Investigator of a grant from the Water Research Resources Center at the University of Massachusetts to investigate the effects of elevated levels of sodium in drinking water on the health of community residents. January 1977 ($4,500).

Co-Principal Investigator. Massachusetts Department of Environmental Protection. Determination of heavy metal background levels. June 1997 ($30,000).

Co-principal Investigator on a contract from the Environmental Protection Agency to conduct: (1) a study of the incidence of death from circulatory system causes between two communities with markedly different sodium levels in drinking water and (2) an analysis of the difference in drinking water quality with respect to minerals and heavy metals between these two communities. July 1977 ($10,000).

Co-principal Investigator on a grant from the U.S. EPA to conduct a study on the effects of chlorine dioxide on mice with low levels of glucose-6-phosphate dehydrogenase in their red cells. October 1977($50,000).

Principal Investigator of a grant from the University of Massachusetts Graduate Research Council - Biomedical Effects Section - to continue studies on the effects of ozone on mice with low levels of glucose-6-phosphate dehydrogenase in red cells. December 1976 ($5,000).

VI. CONSULTING ACTIVITY – Partial Listing

Occupational Health and Safety Administration (OSHA). Advisor and expert witness on litigation proceedings on the area of establishing health risk to workers in different occupations with particular emphasis on chemical coordinating exposure. Consultation has focused on carcinogenic risk from exposure to aromatic amines such as 3,3'-dichlorobenzidine and "MOCA."

Environmental Protection Agency (EPA). (1) Invited as a consultant to advise what EPA's

research priorities should be for FY 1981. (2) Selected to critically review the development of several criteria documents for drinking water contaminants (i.e., antimony, copper, cyanide, dichlorobenzidine, nickel, and zinc). (3) Selected for a national committee to evaluate the methodology by which EPA develops health criteria from which national drinking water regulations are established. (4) Selected as a member of the solvent taskforce to assess risk to the general public from drinking water with variable levels of contamination from a variety of common solvents. (5) Invited member of a select committee to advise EPA on developing methodologies for dealing with epigenetic carcinogens. (6) Selected to chair the health effects committee on nationwide public hearings on volatile organic contaminants in drinking water. (7) Selected as a member of an advisory group to help establish methodologies for assessing risk from carcinogens in drinking water. (8) Selected by EPA to give the principal address on health effects of drinking water pollutants at four nationwide workshops concerning the re-evaluation of the Primary Drinking Water Standards. (9) Selected by EPA to Chair a congressionally mandated study on the comparative health risks of seven different drinking water treatment technologies, (10) consultant Scientific Advisory Board (SAB) on dioxin and environmental exposures.

National Semi-Conductor Co. (Danbury, CT). Provide direction for the development of a new industrial hygiene program. Supervised the developments of risk assessment resulting from occupational exposure to arsenic, arsine, silver, gold, antimony, boron compounds, phophene, hydrofluoric acid, acetic acid, silane, and hydrazine.

North Atlantic Treaty Organization (NATO). Drinking Water and Human Health committee.

Massachusetts State Pesticide Board. Human health effects advisor to an advisory committee of the board. 1977-1981. In September 1981, invited to the State Pesticide Board by the Governor for a 4-year term, but declined invitation.

Ecology and Environment, Inc. (Buffalo, NY). This is an international consulting firm concerned with toxic substance regulation, hazardous wastes, and occupational health. I served on a health advisory board, which provides direction for their industrial hygiene program.

Department of Environmental Quality Engineering (DEQE) for the State of Massachusetts. (1) On matters pertaining to ambient air quality standards and toxic substances in drinking water. (2) Helped to create a 25-hour course on toxicology and risk assessment for DEQE staff. I co-instructed the course. (3) Ad Hoc Committee on sodium in drinking water. (4) Member of a committee to develop a statewide air toxic program.

State of California - Energy Resources Conservation and Development Commission. Provided information on human high-risk groups in a power plant setting.

U.S. Army - Division of Environmental Health and Safety (Fort Dietrick, MD). Provided guidance on the development of a program to establish permissible exposure limits to chemicals employed in various army occupations.

National Sanitation Foundation. Nominated and elected to the NSF Council of Public Health Consultants from 1980 to 1983, specializing in toxicology.

Governor's Hazardous Waste Siting Council. Advise the Massachusetts Legislature and the Governor on the public health considerations in dealing with the proper disposing of hazardous wastes in Massachusetts.

Mitre Corporation. Served on a selected committee to formulate and review methodology for establishing acceptable exposures to toxicants to U.S. Army personnel in combat and training operations.

State of Massachusetts - Department of Public Health and Department of Environmental Quality Engineering Joint Advisory Committee on Environmental Risk Assessment.

National Academy of Sciences. (1) Advised on the development of a possible national study of persons at increased risk to environmental pollutants and (2) Participated as a member of the Safe Drinking Water Committee.

Praeger Scientific Publishers (NY). Reviewer of book proposals in the areas of environmental and occupational health and toxicology.

John Wiley and Sons, Publishers (NY). Reviewer of proposed books in the area of environmental and occupational health and toxicology.

MacMillan Publishing Co. (NY). Reviewer of proposed books in the areas of environmental and occupational health and toxicology.

Sybron Corporation (Rochester, NY). To direct a human risk assessment of exposure to propylene dichloride.

Perkins-Jordan, Co. (Portland, ME). Environmental/industrial engineering company advisor in the area of toxicity of hazardous substances.

Office of Technology and Assessment for the U.S. Congress. I am advising in the area of genetic susceptibility to pollutants.

Pierce, Atwood et al. - a Portland, Maine Law Firm. I am advising with regard to risk assessment for environmental agents.

Canal Electric Co. To advise on the possible health risks of switching from 2.2% sulfur oil to 2.8% sulfur oil for the generation of electricity.

Research Foundation of the American Water Works Association. To develop and conduct courses on toxicology and environmental risk assessment.

Northeast States for Coordinated Air Use Management (NESCAUM). I have been invited to present lectures for NESCAUM staff members on high-risk groups and standard setting during their Air Pollution Health Effects Course. January 1981 (Hartford, CT); March 1982 (Durham, NH).

U.S. Consumer Product Safety Commission and their contractor, JRB Associates. To advise and critically review their studies on consumer products and high risk groups especially children.

Electric Power Research Institute. I have been invited to participate in their nationwide study on the human health effects of inhalable particles from coal-fired power plants.

Gordon A. Enk and Associates, Inc. (Medusa, NY). I was invited to advise in the area of development of toxicological assays to prevent potential human health effects for coal-fired power plants.

Geomet. Inc. (Rockville, MD). I have advised on projects dealing with toxicological hazards in the utility industry.

American Industrial Hygiene Association. Non-Traditional Shiftwork Periods Ad Hoc Committee Membership. July 1982.

Bioassays, Inc. (Woburn, MA). I have advised in the area of developing animal models for predicting the response of humans to ozone and nitrogen dioxide.

Arthur D. Little Company. I have advised on projects dealing with the role of high-risk groups in establishing ambient air standards for mobile source pollutants.

Dynamic Corporation. I advise on a project dealing with assessing the toxicological health hazards associated with the generation of electricity.

Waste Management of Wisconsin, Inc. I advise on the health effects of groundwater contamination by organic substances.

Committee on Human Health Effects and Drinking Water for the American Water Works Association.

Center for Environmental Health and Human Toxicology. Advised on the health effects of formaldehyde.

Massachusetts Railroad Association. To advise on the potential human health risks associated with herbicide spraying.

Harvard University. I advise on the carcinogenic potential of diesel emissions from power generating plants.

State of Florida. I advise the State's Department of Environment on development of a water reuse policy.

City of Los Angeles, Department of Water and Power. I advise concerning risk assessment of carcinogens in drinking water.

State of Connecticut, Preventable Diseases Division. I advise on several areas of health hazards assessment of a wide range of pollutants.

National Institute of Environmental Health Sciences. Selected for the Third Task Force for Research Planning on the Environmental Health Sciences - specialty: Role of host variations, 1984.

American Industrial Health Council. I have advised on the areas of risk assessment and in developing ways to improve scientific communication with the media.

Envirologic Data. I advise in the general area of toxicology and risk assessment.

Academy of Toxicological Sciences. Selected to peer-review the applications of those persons seeking to become board certified in toxicology.

National Science Foundation (NSF). I advise on the area of long-term environmental health research goals with particular emphasis on human high-risk groups and risk assessment.

Council for Environmental Quality (CEQ). I advise on the area of long range planning of EPA research goals as they pertain to pollutant effects on high-risk groups and research methodologies.

U.S. Forestry Service. I advise on the human health risk associated with the aerial spraying of selected pesticides.

U.S. Consumer Product Safety Commission. I was selected based on a national competition to serve as a member of the Consumer Product Safety Commission's Chronic Hazard Advisory Panel on the use of the plasticizer, di(2-ethylhexyl)phthalate (DEHP) in children's products, e.g., pacifer, rubber pants, etc.

Scientific Advisory Panel. Health and Human Services, State of Connecticut.

Media Training. I was one of three toxicologists who participated in an intensive media training program which focused on how to be interviewed by the media on environmental issues. This was sponsored by Chemlawn Inc. February 1985; I had another media training session in November 1985 sponsored by Hoffman-LaRoche, Inc.

Doctor's Data. I was invited to be on the Scientific Board of Directors of this organization. February 1985.

National Academy of Sciences. I was appointed to a special study committee commissioned to assess the health effects of pollutants in commercial aircraft. 1985 to 1986.

World Health Organization. I was invited to participate in development of basic research needs associated with toxic oil syndrome on June 27-28, 1985, in Copenhagen.

Associated Industries of Vermont. I advised on the toxicological basis of the proposed State of Vermont air toxics program.

Gulf and Western, Inc. I advise on the toxicological effects of cadmium and lead contamination of water, air and soil.

State of California - U.S. EPA. I advise on the development of methodologies for establishing a health-based air toxics program.

Rohm and Haas, Inc. I was invited to provide a one-day program on animal extrapolation and risk assessment; also, I was invited to critique their approaches for deriving air quality standards for air toxics.

Southern California Edison. I advise on the environmental and public health implications of soils contaminated with petroleum products.

Monsanto. I was selected to be a member of an expert independent panel of scientists to review toxicology data of pesticide products.

Navy. I advise the Navy on the health effects of contaminants in drinking water.

Syntex Corporation. I advise on the health effects of soil contamination with various organic contaminants.

Tambrands, Inc. I have been invited to become a member of their Institutional Review Committee.

Pacific Power and Light. I have advised in the area of assessing public health implications of PCB contaminated soil.

Digital Equipment Corporation. Assess the health implication of ozone emissions from manufactured equipment.

U.S. Justice Department. Advise on health risk assessment associated with hazardous waste sites.

Department of Defense, U.S. Army. Advise on the extrapolative relevance of alternative animal models for predicting human responses to environmental toxins.

Council for Agricultural Science and Technology. Invited to serve on national committee to assess risk from 2-4D exposure.

Alliance Technologies. Advise in the area of risk assessment and toxicology on a variety of environmental issues.

Roy Weston, Inc. Advise in the area of risk assessment and toxicology.

Colorado Department of Public Health. Advised on the development of risk assessment methodologies to estimate human health risks from possible exposure from the Rocky Mountain Arsenal.
NOITE Corporation. Denver, Colorado. Advise on the potential public health risks associated with drinking water contaminants.

Smith, Kline and Beckman. Advise on the public health risks associated with incineration of medically related waste.

Gelman, Inc. Advise on the public health implications of organic contaminants in groundwater.

GZA Corporation. Advise on the public health risks of petroleum contamination.

Gelman Sciences. Advise on the public health risk of various issues relating to risk assessment procedures to estimate public health hazards for chemical contaminants such as 1,4 dioxane.

State University at Albany - Center for Policy Research. Advise on the issue of medical infectious waste and public health.

World Health Organization (WHO). I advise on the role of genetic factors in affecting the occurrence of occupationally-induced disease.

Woodward-Clyde Consultants, Inc. Advise on the public health risks associated with exposure to toxics from multi-media.

Environ Corp. Advise on the issue of soil ingestion by children.

W.R. Grace. Advise on various risk assessment issues.

Committee on Urban Environmental Protection for the Division of Urban Affairs of the National Association of State Universities and Land Grant Colleges.

Member of the International Joint Commission, Great Lakes Science Advisory Board's Health Committee, 1991-1992.

Florida Power and Light. Advise on various risk assessment areas.

3M Corporation. Advise on environmental and occupational health issues.

National Academy of Sciences. Invited to be a member of the committee assessing the human health effects of the fuel additive MTBE.

State of Colorado. Advised on risks associated with contamination at the Rocky Mountain Arsenal. 1988-present (2002).

Journal Reviewer (examples of):

2014-2015

- ACS Central Science
- BBA-Molecular Cell Research
- Cancer Research
- Chemico-Biological Interactions
- Ecotoxicology and Environmental Safety
- Environmental Research
- Environmental Toxicology & Chemistry
- Environmental Toxicology & Pharmacology
- Human and Experimental Toxicology
- International Journal Plant Biology
- Neuro Toxicology
- Plant Disease
- Plant Physiology
- PLOS One
- Proteomics
- RAD 2015 Proceedings
- Toxicological Sciences

Past Years

- Ageing Research Reviews
- Archives of Environmental Contamination and Toxicology
- Biogerontology
- BioEssays
- BioMed Central Genomics
- Chemical Research in Toxicology
- Chemosphere
- Drug Safety
- Ecology Letters

Ecotoxicology
Ecotoxicology and Environmental Safety
Environment International
Environmental and Experimental Botany
Environmental Health Perspectives
Environmental Science and Technology
Experimental Gerontology
Free Radical Biology and Medicine
Fresenius Environmental Bulletin
Food and Chemical Toxicology
Frontiers in Bioscience
GLIA
Hazarouds Materials
HortScience
Human and Experimental Toxicology
International Journal of Obesity
International Journal of Toxicology
Italian Journal of Zoology
Journal of Alzheimer's Disease
Journal of Plant Growth Regulation
Journal of Zhejiang University Biologia Plantarum
Journal of Zoology
Molecular Biology Reports
Neuro Toxicology
Pest Management Science
Plant Physiology
Rejuvenation Research
Risk Analysis
Science
Science of the Total Environment
Toxicology Sciences

Journal Editorship:

Editor-in-Chief - Dose Response (formerly Non-linearity in Biology, Toxicology and medicine), 2005-present
Guest-Editor – Proceedings of the National Academy of Science
Advisory Board - Invited member of the Advisory Board of the ICCNS Journal of Cell Communications and Signaling, 2012
Editor-in-Chief – Non-linearity in Biology, Toxicology, and Medicine, 2001-2005
Editor-in-Chief - Human and Ecological Risk Assessment - 1995-2009
Editorial Board - Inhalation Toxicology - 1990-1998

Editorial Board - Soil and Sediment Contamination: An International Journal – 1993-2000
Editorial Board - Human and Experimental Toxicology - 1995-present
Editorial Board - Environmental Toxicology and Safety - 1994-1998
Editorial Board - Biomedical and Environmental Sciences - 1996-1998

Book Editorship:

Guest Editor, Distribution of Artificial Radionuclides in the Abandoned Cattle in the Evacuation Zone of the Fukushima Daiichi Nuclear Power Plant. Proceedings of the National Academy of Sciences. 2012.

Co-Editor, Hormesis: A Revolution in Biology, Toxicology and Medicine. Humana Press Inc., 2010, 213 pages.

Advisory Board, Toxicology Desk Reference, The Toxic Exposure and Medical Monitoring Index, 1996.

Co-Editor, Annual review of Ecotoxicology and Environmental Toxicology & Chemistry, 1996.

Co-Editor, Current Topics in Ecotoxicology and Environmental Chemistry, published by Taylor and Francis, 1995-present.

Editor of the series Environmental Health and Toxicology published by Lewis Publishers, 1990-1993.

Co-Editor of a Monograph Series on Remedial Technologies for Hydrocarbon Contaminated Soils published by Lewis Publishers, 1990-1992

Co-Editor, Soils Contaminated by Petroleum, Environmental and Public Health Effects, John Wiley & Sons, 1988

VII. ACADEMIC AND OTHER HONORS

Invited member of the Advisory Committee of the Nuclear Safety & Security Commission (Project #1501007) in Korea
Awarded the Petr Beckmann Award by Doctors for Disaster Preparedness for courage and achievement in defense of scientific truth and freedom.
Honorary Degree, Doctor of Science. School of Nursing and Medical Radiation Sciences Program, McMaster University Canada
Invited member of the Advisory Board of the ICCNS Journal of Cell Communications and Signaling, 2012
Honorary member of the International CCN Society, 2012
Awarded the second International Cell Communication and Signaling-Springer award, Belfast

Northern Ireland, 2010.
Awarded the Marie Curie Prize from the World Council of Nuclear Workers at the 8th LOWRAD International Conference in Rio de Janeiro, Brazil, 2010.
Selected to present the Third Annual Environmental Toxicology Lectureship at the Institute for Environmental Studies at the University of Illinois, 1991.
Nominated for Teacher of the Year Award - several times
Appointed to the Food and Nutrition Board of the National Research Council, 1988- 1991.
Appointed by the Institute of Medicine to the Food and Nutritional Board, 1988-1990.
Adrian Rondileau Award for outstanding leadership and professional achievement, 1988.
Appointed to the National Academy of Sciences Safe Drinking Water Committee, 1982-1984, 1986.
Appointed to the NATO countries Safe Drinking Water Committee
Appointed to the 11 member Scientific Counselors of the Agency for Toxic Substances and Disease Registry
Phi Delta Phi - a national academic fraternity
Kappa Delta Pi - a national education fraternity requiring the member to be in the upper 1/10 of the graduating class.
William Vinal Zoological Award - awarded to graduating senior biology major with the highest academic average in zoology.
Danforth Fellowship Nomination
Dean's List - 7 semesters

VIII. SOCIETIES

International Dose-Response Society, 2003-present
Association for the Advancement of American Sciences (AAAS)
Society for Occupational and Environmental Health (SOEH) - Elected to the Governing Council, 1980-1982.
American College of Toxicology (ACT) - Elected to be a Councilor, 1981-1983
Society of Environmental Toxicology and Chemistry (SETAC)
Society of Risk Analysis (SRA)
Society of Toxicology (SOT)
New England Chapter of the Society of Toxicology - Councilor
International Society for the Regulatory Toxicology and Pharmacology
Council for Health and Environmental Safety of Soil (CHESS) – Selected to Chair, 1987-1997
BELLE, Chairman of the Advisory Committee, 1990-present.

IX. UNIVERSITY ASSIGNMENTS

School of Health Sciences and Environemtnal Health Sciences - Personnel Committee, 2006-2009, 2011-2015
Environmental Health Sciences Department – Personnel Committee, 2015
Animal Care

University-wide Environment Committee
Advisory Board of the Institute of Environmental Studies
Ph.D. Policy and Admissions Committee
Biohazards Regulation and Control Committee
Advisory Board of the Water Research Resource Center
Division of Public Health - Nutrition Department Joint Student Admission Committee
Search Committee for New Director of the Division of Public Health, 1983
Teaching Evaluation Committee, 1982
By-Laws Committee, 1978-1982
Curriculum Committee, 1978-1981
Academic Affairs Council, 1976-1979
Ph.D. Proposal Committee, 1977-1978

X. CERTIFICATION

Elected to the Board of Directors of the Academy of Toxicological Sciences,1987-1989
Elected Vice President of the Board of Directors of the Academy of Toxicological Sciences, 1987-1989
Board Certified in General Toxicology by the Academy of Toxicological Sciences,1982, renewed 1987- 2007, 2012-2017
Elected to the Professional Evaluation Board

XI. VISITING PROFESSORSHIP

Visiting Professor Lecture Program, September 2011. U.S. Food and Drug Administration, White Oaks Campus, Silver Spring, Maryland.

University of Illinois at Champagne-Urbana, April 1989. Toxicology scholar in residence. Invited to present seminar/lecture on toxicology and human risk assessment.

Harvard University, School of Public Health, September 1985, and 1986. Invited to be a guest faculty member in the course "Risk Analysis in Environmental Health." My topic is "Use of Animal and Other Data as Predictors of Human Risks."

University of North Carolina School of Public Health at Chapel Hill. February 12-16, 1984.

XII. PUBLICATIONS

2018

Calabrese EJ. (2018). Was Muller's 1946 Nobel Prize research for radiation-induced gene mutations peer-reviewed? PEHM (submitted).

Calabrese EJ. (2018). From Muller to mechanism: How LNT became the default model for cancer risk assessment. Environ Poll (submitted).

Calabrese EJ. (2018). The linear no-threshold (LNT) dose response model: A comprehensive assessment of its historical and scientific foundations. Crit Rev Toxicol (submitted).

Agathokleous E, Kitao M, Calabrese EJ. (2018). Biphasic effect of abscisic acid on plants: a hormetic viewpoint. Botany (submitted)

Kozumbo WJ, Leak RK, Calabrese EJ, Johnson TE, Mitchell JR, Ozaki CK, Wetzker R, Anderson ME, Bast A, Belz RG, Botker HE, Koch S, Mattson MP, Gidday JM, Simon RP, Jirtle RJ. (2018). Enhancing the amplitude and duration of hormesis-induced resilience. Workshop summary, October 2017. Progress in Neurobiology (submitted).

Calabrese EJ. (2018). The additive to background assumption in cancer risk assessment: A reappraisal. Environ Res (submitted).

Agathokleous E, Kitao M, Ristow M, Mattson MP, Calabrese EJ. (2018). Environmental hormesis and its fundamental biological base rewrite the history of toxicology. Environmental Research (accepted)

Agathokleous E, Kitao M, Calabrese EJ. (2018). The concept of environmental hormesis can advance the current scientific base of global change biology. Global Change Biology (submitted).

Calabrese EJ. (2018). Regulation of carcinogens and chemicasl: What went wrong. In: Science and Liberty (PJ Michaels, T Kealey, editors), Chapter 8 Cato Institute (submitted).

Agathokleous E, Belz RG, Calabrese EJ, Clatayud V, De Marco A, Hoshika Y, Kitao M, Saitanis CJ, Sicard P, Paoletti E. (2018). Predicting the effect of ozone on vegetation: A comparison of the linear non-threshold (LNT). (submitted)

Calabrese EJ. (2018). The dose-response revolution: How hormesis became significant. An Historical and Personal Reflection. In: The science of hormesis in health and longevity (S Rattan and M Kyriazis, Editors). Elsevier Publishers (in press).

Calabrese EJ. (2018). Using preconditioning to build biological shields: A novel approach for

enhancing resilience to toxic agents, traumatic illness/injury and age-related degenerative diseases. In: Chemical Warfare Agents (H. Salem, B. Lukey, editors). CRC Press (in press).

Calabrese EJ, Ricci PF. (2018). How hormesis will change the risk assessment process. Encyclopedia of Environmental Health, 2nd edition. Elsevier Publishers.

Agathokleous E, Kitao M, Calabrese EJ. (2018). Emission of volatile organic compunds (VOCs) from plants shows a biphasic pattern within a hormetic context. Environ Poll (in press).

Agathokleous E, Kitao M, Calabrese EJ. (2018). The rare earth element (REE) lanthanum (La) induced hormesis in plants. Environ Poll (in press).

Calabrese EJ, Rubio-Casillas A. (2018). Biphasic effects of THC in memory and cognition. European Journal of Clinical Investigation 2018;e12920.

Calabrese V, Santoro A, Salinaro AT, Modafferi S, Scuto M, Albouchi F, Monti D, Giordano J, Zappia M, Franceschi C, Calabrese EJ. (2018). Hormetic approaches to the treatment of Parkinson's Disease: Perspective and possibilities. Journal of Neuroscience Research (in press).

Iavicoli I, Leso V, Fontana L, Calabrese, EJ. (2018). Nanoparticle exposure and hormetic dose-responses: An update. International Journal of Molecular Sciences (in press).

Calabrese EJ, Iavicoli I, Calabrese V, Cory-Slechta DA, Giordano J. (2018). Elemental mercury neurotoxicity and clinical recovery of function: A review of findings, and implications for occupational health. Environ Res 163:134-148.

Calabrese EJ. (2018). Post-conditioning hormesis creates a "subtraction to background" disease process: Biological, aging, and environmental risk assessment implications. Journal of Cell Communication and Signaling 12:31-34.

Salinaro AT, Pennisi M, DiPaola R, Scuto M, Crupi R, Cambria M, Ontario ML, Tomasello M, Uva M, Maiolino L, Calabrese EJ, Cuzzocrea S, Calabrese V. (2018). Neuroinflammation and neurohormesis in the pathogenesis of Alzheimer's Disease and Alzhemimer-linked pathologies: Modulation by nutritional mushrooms. Immunity & Ageing 15:1-8.

Calabrese V, Santoro A, Monti D, Crupi R, Di Paola R, Latteri S, Cuzzocrea S, Zappia M, Giordano J, Calabrese EJ, Franceschi C. (2018). Aging and Parkinsons's Disease: Inflammaging, neuroinflammation and biological remodeling as key factors in pathogenesis. Free Radical Biology and Medicine 115:80-91.

Hanekamp J, Calabrese EJ. (2018). Risk assessment and analysis: Part I. Risk assessment In: Encyclopedia of Chemical Law – Products, Soil, and Waste of Stanford UP (submitted).

Hanekamp J, Calabrese EJ. (2018). Risk assessment and analysis: Part 2. Decision tools In: Encyclopedia of Chemical Law – Products, Soil, and Waste of Stanford UP (submitted).

2017

Calabrese EJ. (2017). Originator of the hormesis concept: Rudolf Virchow or Hugo Schulz. Hum Exp Toxicol doi: 10.1177/096032711775-1237 (Epub ahead of print).

Calabrese EJ, Lehr J. (2017). The final demise of the linear no threshold (LNT) theory. Environ Clim News. Vol 20, Number 4.

Calabrese EJ. (2017). The mistaken birth and adoption of LNT: An abridged version. Dose-Response 2017:1-3.

Calabrese EJ, Mattson MP. (2017). How does hormesis impact biology, toxicology and medicine? Aging Mech Dis 3:13.

Calabrese EJ. (2017). Perspectives on hormesis and implications for pesticides. In: Pesticide Dose: Effects on the Environment and Target and Non-Target Organisms, Chapter 7 (SO Duke, P Kudsk, K Solomon, Editors). ACS Symposium Series, American Chemical Society 1249:83-100.

Calabrese EJ. (2017). Flaws in the LNT single-hit model for cancer risk: An historical assessment. Environ Res 158:773-788.

Calabrese V, Giordano J, Crupi R, Di Paola R, Ruggieri M, Bianchini R, Ontario ML, Cuzzocrea S, Calabrese EJ. (2017). Hormesis, cellular stress response and neuroinflammation in schizophrenia: Early onset versus late onset state. J Neurosci Res 95(5): 1182-1193.

Pennisi M, Crupi R, Di Paola R, Ontario ML, Bella R, Calabrese EJ, Crea R, Cuzocrea S, Calabrese V. (2017). Inflammasomes, hormesis, and antioxidants in neuroinflammation: Role of NRLP3 in Alzheimer disease. J Neurosci Res 95(7): 1360-1372.

Calabrese EJ. (2017). Hormesis commonly observed in the assessment of aneuploidy in yeast. Environ Poll, 225:713-728.

Calabrese EJ. (2017). Obituary notice: LNT dead at 89 years, a life in the spotlight. Environ Res 155:176-178. http://dx.doi.org/10.1016/j.envres.2017.02.031

Calabrese EJ, Calabrese V, Giordano J. (2017). The role of hormesis in the functional performance and protection of neural systems. Brain Circul 3:1-13.

Calabrese EJ. (2017). LNTgate: The ideological history of cancer risk assessment. Toxicol Res Appl 1-3; DOI: 10.1177/2397847317694998.

Calabrese EJ. (2017). The threshold vs LNT showdown: Dose rate findings exposed flaws in the

LNT model. Part I. The Russell-Muller debate. Environ Res 154:435-451.

Calabrese EJ. (2017). The threshold vs LNT showdown: Dose rate findings exposed flaws in the LNT model. Part 2. How a mistake led BEIR I to adopt LNT. Environ Res 154:452-458.

Giordano J, Bikson M, Kappenman ES, Clark VP, Coslett HB, Hamblin MR, Hamilton R, Jankord R, Kozumbo WJ, McKinley RA, Nitsche MA, Reilly JP, Richardson J, Wurzman R, Calabrese EJ. (2017). Mechanisms and effects of transcranial direct current stimulation (tDCS). Dose Response 1-22; DOI:10.1177/1559325816685467.

Nascarella MA, Calabrese EJ. (2017). Hazard assessment and the evaluation of rare earth element dose-response relationships. In: Rare Earth Elements in Human and Environmental Health: At Crossroads between Toxicity and Safety (Pagano G, Editor). Chapter 8, Pan Stanford Publishing Pte Ltd., pp 183-194.

Calabrese EJ, Dhawan G, Kapoor R. (2017). Radiotherapy for pertussis: An historical Assessment. Dose-Response 15(2): May 8.

Janiak MK, Wincenciak M, Cheda A, Nowosielska EM, Calabrese EJ. (2017). Cancer immunotherapy: How low-level ionizing radiation can play a key role. Cancer Immunology, Immunotherapy 66(7): 819-832.

Wang D, Calabrese EJ, Lian B, Lin Z, Calabrese V. (2017). A pharmacological rosetta stone: Hormesis as a mechanistic approach to understanding and describing herbal treatments of traditional Chinese medicine within a Western biomedical framework. Pharmacology and Therapeutics Epub 2017 Nov 8.

Calabrese EJ. (2017). Societal threats from ideologically driven science. Acad Quest J 30(4):405-418.

Calabrese EJ. (2017). A glance into how the cold war and governmental loyalty investigations came to affect a leading US radiation geneticist: Lewis J. Stadler's nightmare. Philos Ethics Human Med. 12:8.

Calabrese EJ. (2017). Hormesis and homeopathy: A step forward. Homeopathy 106:131-132.

Sun H, Calabrese E, Zheng M, Wang D, Pan Y, Lin Z, Liu Y. (2017). A swinging seesaw as a novel model mechanism for time-dependent hormesis under dose-dependent stimulatory and inhibitory effects: a case study on the toxicity of antibacterial chemicals to Aliivibrio fisheri. Arch Toxicol (submitted).

Calabrese V, Calabrese EJ, Carare RO, Cedazo-Minguez A, Frenkel D, Korczyn A, Popescu BO. (2017). Major pathogenic mechanisms in vascular cognitive impairment. BMC Med Manus (In Press).

Calabrese EJ, Nascarella MA. (2017). Hormesis: accessing low dose exposure to chemical warfare agents. In: Chemical Warfare Agents (tentative title). Chapter 12. (submitted).

Shamoun DY, Calabrese EJ. (2017). On objective risk. Risk Research (submitted).

Calabrese EJ. (2017). The linear no-threshold (LNT) dose response model: A comprehensive assessment of its historical and scientific foundations. Critical Reviews in Toxicology (submitted).

Calabrese V, Franceschi C, Aurelia S, Monti D, Calabrese EJ. (2017). Therapeutic strategies in the prevention and treatment of Parkinsons's Disease via hormesis. Journal Neurosci Res (Submitted)

Calabrese EJ. (2017). Scientific foundations of LNT challenged. (In Prep).

Shamoun DY, Calabrese EJ. Williams R, Broughel. (2017). The case against LNT. Mercatus Center at George Mason University. Arlington VA. Risk Analysis (In Prep).

2016

1. Calabrese EJ, Dhawan G, Kapoor R, Iavicoli I, Calabrese V. (2016). Hormesis: A fundamental concept with widespread biological and biomedical applications. Gerontology 62(5):530-535.

2. Calabrese V, Giordano J, Ruggieri M, Berritta D, Trovato A, Ontario ML, Bianchini R, Calabrese EJ. (2016). Hormesis, cellular stress response, and redox homeostasis in autism spectrum disorders. J Neurosci Res 94:12(SI):1488-1498.

3. Calabrese V, Giordano J, Signorile A, Ontario ML, Castorina S, De Pasquale C, Eckert G, Calabrese EJ. (2016). Major pathogenic mechanisms in vascular dementia: Role of cellular stress response and hormesis in neuroprotection. J Neurosci Res 94(SI12):1588-1603.

4. Calabrese EJ. (2016). The emergence of the dose-response concept in biology and medicine. Intern J Mol Sci 17(12): Article Number UNSP 2034.

5. Calabrese EJ. (2016). LNTgate: How scientific misconduct by the US NAS led to governments adopting LNT for cancer risk assessment – Rebuttal to Letter of Beyea (2016). Environ Res 148:535-546.

6. Calabrese EJ. (2016). Pre- and post-conditioning hormesis in elderly mice, rats, and humans: Its loss and restoration. Biogerontology 17(4):681-702.

7. Mollereau B, Rzechorzek NM, Roussel Bd, Sedru M, Van denBrink D, Bailly-Maitre B, Palladino F, Medinas DB, Domingos PM, Hunot S, Chandran S, Birman S, Baron T, Vivien D, Duarte CB, Ryoo HD, Steller H, Urano F, Chevet E, Kroemer G, Ciechanover A, Calabrese EJ, Kaufman RJ, Hetz C. (2016). Adaptive preconditioning in neurological diseases - Therapeutic insights from proteostatic perturbations. Brain Res 1648(SI):603-616.

8. Calabrese EJ, Shamoun DY, Hanekamp JC. (2016). The integration of LNT and hormesis for cancer risk assessment optimizes public health protection. Health Physics 110(3):256-259.

9. Calabrese EJ. (2016). Preconditioning is Hormesis. Part I. Documentation, dose-response features and mechanistic foundations. *Pharm Res* 110:242-264.

10. Calabrese EJ. (2016). Preconditioning is Hormesis. Part II: How the conditioning dose mediates protection. Dose optimization within temporal and mechanistic frameworks. Pharm Res 110:265-275.

11. Calabrese EJ. (2016). Model uncertainty via the integration of hormesis and LNT as the default in cancer risk assessment. Dose-Response 2015:1-5. DOI: 10.1177/1559325815621764.

12. Jonas WB, Calabrese EJ. (2016). Learning from the history of integrative preventive medicine to address our current health care challenges. In: Textbook of Integrative Preventive Medicine (R Carmona, M Liponis, Editors). Oxford Press.

13. LNTgate: How scientific misconduct by the US NAS led to governments adopting LNT for cancer risk assessment. Environ Res 148:535-546.

2015

1. Calabrese EJ. (2015). Hormesis: Principles and applications. Homeopathy 104(2):69-82.

2. Calabrese EJ. (2015). Historical foundations of hormesis. Homeopathy 104(2):83-89.

3. Calabrese EJ. (2015). Hormesis within a mechanistic context. Homeopathy 104(2):90-96.

4. Calabrese V, Scapagnini G, Davinelli S, Koverech G, Koverech A, De Pasquale C, Sallinaro AT, Scuto M, Calabrese EJ, Genazzani AR. (2015). Sex hormonal regulation and hormesis in aging and longevity: Role of vitagenes. J Cell Comm Sign 8:369-384.

5. Calabrese EJ. (2015) On the origins of the linear no-threshold (LNT) dogma by means of untruths, artful dodges and blind faith. Environ Res 142:432-442.

6. Calabrese EJ, Shamoun DY, Hanekamp JC. (2015). Cancer risk assessment: Optimizing human health through linear dose-response models. Fd Chem Toxic 81:137-140.

7. Calabrese EJ, Dhawan G, Kapoor R, Iavicoli I, Calabrese V. (2015). What is hormesis and its relevance to healty ageing and longevity. Biogerontology 16:693-707.

8. Calabrese EJ, Dhawan G, Kapoor R. (2015). The use of X rays in the treatment of bronchial asthma: A historical Assessment. Rad Res 184:180-192.

9. Calabrese V, Datilo S, Petralia A, Parenti R, Pennisi M, Koverech G, Calabrese V, Graziano A, Monte I, Maiolino L, Ferreri T, Calabrese EJ. (2015) Analytical approaches to the diagnosis and treatment of aging and aging-related disease: Redox status and proteomics. Free Rad Res 49:511-524.

10. Dattilo S, Mancuso C, Koverech G, Di Mauro P, Ontario ML, Petralla CC, Petralla A, Maiolino L, Serra A, Calabrese EJ, Calabrese V. (2015). Heat shock proteins and hormesis in the diagnosis and treatment of neurodegenerative disease. Immun Aging 12:20 DOI:10.1186/s12979-015-0046-8.

11. O'Connor MK, Calabrese EJ. (2015). Response to Comments on "Estimating Risks of Low Radiation Doses—A Critical Review of the BEIR VII Report and its Use of the Linear No-Threshold (LNT) Hypothesis" Rad Res 183:481-484.

12. Hanekamp JC, Bast A, Calabrese EJ. (2015). Nutrition and health – transforming research traditions. Crit Rev Food Sci Nutr, 55(8):1074-1080; doi: 10.1080/10408398.2012.680525.

13. Calabrese EJ. (2015). The dose response: Comparing hormesis and threshold models. In: Fundamentals of Ecotoxicology. The Science of Pollutions, 4th Edition (MC Newman, editor). CRC Press, Boca Raton, FL, pp 654.

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1978

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1977

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1975

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1974

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1973

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1972

1. Calabrese, E.J. (1972). The effects of phosfon on the growth of *Mentha piperita* L. Master's thesis at State College at Bridgewater, Massachusetts.

1968

1. Calabrese, E.J. (1968). The effects of a phosphon on *Mentha piperita* L. in different growth media. Eastern College Science Conference. Yale University, New London, CT. April 20th.

XIII. PRESENTATIONS AT MAJOR CONFERENCES/INVITED SEMINARS

2018

Cottrell M, Mills W, Calabrese EJ. Funding trends in hormetic research. Climate Leaderhsip Summit. Univeristy of Massachusetts, Amherst MA. April 8, 2018.

Kozumbo WJ, Leak RK, Calabrese EJ, and 13 other co-authors. Enhancing the amplitude and duration of hormesis-induced resilience: Workshop summary October 2017. The 17th Annual

International Dose-Response Conference. Preconditioning: in Biology and Medicine. Mechanisms and Translational Research. Univeristy of Massachusetts, Amherst MA. April 17-18, 2018.

Agathokleous E, Kitao M, Calabrese EJ. Lanthanum induces hormesis in plants: A perspective for agronomy. The 17th Annual International Dose-Response Conference. Preconditioning: in Biology and Medicine. Mechanisms and Translational Research. Univeristy of Massachusetts, Amherst MA. April 17-18, 2018.

Agathakleous E, Calabrese EJ, and 8 other co-authors. Hormesis for predicting the effect of ozone on vegetation. The 17th Annual International Dose-Response Conference. Preconditioning: in Biology and Medicine. Mechanisms and Translational Research. Univeristy of Massachusetts, Amherst MA. April 17-18, 2018.

Cottrell M, Mills W, Calabrese EJ. Funding trends in hormetic research. The 17th Annual International Dose-Response Conference. Preconditioning: in Biology and Medicine. Mechanisms and Translational Research. Univeristy of Massachusetts, Amherst MA. April 17, 2018.

Calabrese EJ. Linear no-threshold (LNT) dose-response and what it means to you. University of Rhode Island, Kingston RI. April 6, 2018.

Calabrese EJ. Hormesis: How it can improve public health and medicine. Bridgewater State University, Bridgewater MA. April 5, 2018.
Calabrese EJ. Linear no-threshold (LNT) dose-response and what it means to you. Bridgewater State University, Bridgewater MA. April 5, 2018

Calabrese EJ. Hormesis: How it can improve public health and Medicince. International Academy of Oral Medicine & Toxicology. Fundamentals of Biological Dentistry. Denver, CO, March 23-24, 2018.

Calabrese EJ. Hormesis: The linear dose response for cancer risk assessment: New findings challenge its scientific foundations and use by regulatory and public health agencies. International Academy of Oral Medicine & Toxicology. Fundamentals of Biological Dentistry. Denver, CO, March 23-24, 2018.

2017

Calabrese EJ. Hormesis: enhancing performance and building biological shields. Defense Advanced Research Projects Agencys (DARPA), Washington, DC. December 20, 2017.

Calabrese EJ. Homeopathy Conference. Hormesis in biology and medicine: Why it needs to be taught in medical school. University of Massachusetts, Amherst, MA. November 11, 2017.

Calabrese EJ. Hormesis in biology and medicine: Why it needs to be taught in medical school. School of Medicine. Georgetown University, Washington DC. November 7, 2017.

Calabrese EJ. Linear no-threshold (LNT) dose-response and what it means to you. MICB 702 Course, Georgetown University, Washington DC. November 6, 2017.

Calabrese EJ. LNT and its history Mount Holyoke College. October 24, 2017.

Calabrese EJ. Conference Moderator. AF Hormesis Conf. October 21 & 22, 2017.

Calabrese EJ. Overview to Air Force Hormesis Workshop. UMass October 21, 2017.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. EHSC Seminar Series, University of Rochester, Rochester NY. September 21, 2017.

Calabrese EJ. The search for truth in regulatory science; How LNT was born and sustained – a story of mistakes, deceptions, and failed public policy. CATO Institute, Washington DC. July 21, 2017.

Calabrese EJ. Hormesis overview. Annual International Conference on Dose-Response: Preconditioning in biology and medicine. Mechanisms and translational research. April 18, 2017.

Dhawan G, Calabrese EJ. Radiotherapy for pertussis: An historical assessment. Annual International Conference on Dose-Response: Preconditioning in biology and medicine. Mechanisms and translational research. April 18, 2017.

Calabrese EJ. Hormesis: what it means for toxicology and risk assessment. Scientific Session, Low-dose non-monotonic responses. Society of Toxicology 56th Annual Meeting and ToxExpo, Baltimore, MD. March 14, 2017.

Calabrese EJ. How LNT was born and sustained - A Story of Mistakes, Deceptions, and Failed Public Policy. MA Department of Public Health. Boston, MA. January 9, 2017

2016

Calabrese EJ. How LNT was born and sustained. A story of mistakes, deceptions, and failed public policy. Toxicology School of Pharmacy - University of Connecticut, Storrs, CT. November 28, 2016.

Calabrese EJ. Dose-response models, hormesis and implications for regulations. Podcast Intervew. Center for Industrial Progress. November 16, 2016.

Calabrese EJ. Hormesis: Its biological foundation and implications for pharmacology, medicine

and public health. American Course on Drug Development and Regualtory Sciences Session 3. Washington DC. November 9, 2016.

Calabrese EJ. Seminar series. Hormesis: Role in biology, medicine, and public health. Environmental Health Sciences, University of Massachusetts, Amherst, MA. November 7, 2016.

Kozumbo WJ, Calabrese EJ. Enhancing biological performance: occurrence, mechanisms and applications. Wright Patterson Air Force Base, Ohio. November 7, 2016.

Calabrese EJ. Hormesis: Role in Biology, Medicine, and Public Health. Institut fur Molekular Zellbiologie, CMB. Jenna, Germany. October 27, 2016.

Calabrese EJ. Hormesis: Role in biology, medicine, and public health. Arnold School of Public Health, University of South Carolina, Columbia, SC. October 21, 2016.

Calabrese EJ. Hormesis: Role in Biology, Medicine, and Public HealthBridgewater State University, Bridgewater, MA. October 14, 2016.

Calabrese EJ. Hormesis: Role in Biology, Medicine, and Public HealthCollege of Nursing, University of Massachusetts, Amherst, MA. October 11, 2016.

Calabrese EJ. How LNT was born and sustained. A story of mistakes, deceptions, and failed public policy. Health Canada, Canada. September 29, 2016.

Calabrese EJ. Hormesis: Role in biology, medicine, and public health. University of Ottawa, Ottawa, ON. September 28, 2016.

Calabrese EJ. Hormesis: Role in biology, medicine, and public health. Canadian Nuclear Laboratories, Petawawa, ON. September 27, 2016.

Calabrese EJ. The road to linearity. Canadian Nuclear Laboratories, Petawawa, ON. September 27, 2016.

Calabrese EJ. Hormesis: Adaptive Responses in Biology and MedicineSociety for Cancer Research and Communication, Department of Radiation Oncology, Dr Balabhai Nanavati Hospital, Vile Parle, Mumbai, India. August 6, 2016.

Calabrese EJ. Linear No Threshold Model. Wall Street Journal Interview. June 28, 2016

Calabrese EJ. Atomic Insights. Interview. June 6, 2016.

Calabrese EJ. Interview. City Univerity, London UK. May 19, 2016.

Calabrese EJ, Dhawan G, Kapoor R. Preconditioning is hormesis Part I: documentation, dose-response features and mechanistic foundations. Presented at the Annual Conference of the

International Dose Response Society. University of Massachusetts. Amherst MA. April 20, 2016.

Calabrese EJ, Dhawan G, Kapoor R. How the conditioning dose mediate protection: dose optimization within temporal and mechanistic frameworks. Presented at the Annual Conference of the International Dose Response Society. University of Massachusetts. Amherst MA. April 20, 2016.

Calabrese EJ. How the US NAS misled the world community on cancer risk assessment. Hartford University. Hartford CT. April 26, 2016.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. Dalhousie University, Faculty of Agriculture. Truro NS, Canada. April 1, 2016.

Calabrese EJ. Preconditioning is hormesis. Dalhousie University, Special Seminar, Community Health & Epidemiology. Halifax NS, Canada. March 31, 2016.

Calabrese EJ. International Life Sciences Institute (North America). Conundrum: How do we define the continuum - from perturbation to adverse effects? Lessons Learned: Hormesis. St. Petersburg, FL. Jan 25-26, 2016.

2015

Shamoun DY, Calabrese EJ. On objective risk. Society for Risk Analysis. Arlington, VA. December 9, 2015

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. Boston College Biology Seminar. Chestnut Hill, MA. November 3, 2015.

Calabrese EJ. The integration of LNT and hormesis for cancer risk assessment optimizes public health protection. Risk Assessment Speciality Section. Reston, VA. October 14, 2015.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. University of Massachusetts, Food Science Department. Amherst, MA. September 23, 2015.

Shamoun DY, Calabrese EJ. On objective risk. The Science, Policy and Risk Forums. ORACBA and National Capital Area Chper of the Society of Risk Analysis at the USDA. Washinton DC. September 15, 2015.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. American Chemical Society AGRO Session. Boston, MA. August 16-20, 2015.

Calabrese EJ. How the US NAS misled the wold community on cancer risk assessment. Doctors for Disaster. July 31-August 1, 2015.

Calabrese EJ. Hormesis: Its scientific foundations and biochemical regulatory applications. Doctors for Disaster. July 31-August 1, 2015.

Calabrese EJ. Introduction to hormesis: Adaptive responses in biology and medicine. Air Force Planning Meeting: Dosimetry and mechanisms mediating response to tDCS. Univeristy of Massachusetts. Amherst MA. July 8 & 9, 2015.

Calabrese EJ. How the US NAS misled the world community on cancer risk assessment. Polymer Science, University of Massachusetts. Amherst MA. 2015.

Calabrese EJ. How the linear dose response became the default model for cncer risk assessment. New England Chapter of the American Association of Physicists in Medicine. Sturbridge, MA. May 29, 2015.

Calabrese EJ. History of LNT. New England Chapter of the Health Physics Society. Westford, MA. May 27, 2015.

Calabrese EJ. How the linear dose response became the default model for cancer risk assessment. Eastern Research Group-Health Effects Institute. Boston MA. May 20, 2015.
Calabrese EJ, Blain R. . Hormetic Mechanism-Receptor/Cell Signaling Pathways. Society of Toxicology. Phoenix, AZ. March, 2015.

2014

Calabrese EJ, Shamoun DY. The case against LNT. Part I: History, origin, and competing evidence. Soceity of Risk Analysis, Denver CO. December, 2014.

Shamoun DY, Calabrese EJ. Guidelines for objective risk assessment practices. Society for Risk Analysis. Denver, CO. December 10, 2014.

Calabrese EJ. Australasian Radiation Protection Society Conference. How the US NAS misled the world community on cancer risk assessment, Hobart Tasmania Australia. October 28, 2014.

Calabrese EJ. Duke University, Integrated Toxicology and Enviornmental Health Program Symposium. Biphasic dose responses in biology, toxicology and medicine. October 24, 2014.

Calabrese EJ. Michigan State University, Department of Pharmacology and Toxicology. How the US NAS misled the world community on cancer risk assessment. September 19, 2014.

Calabrese EJ. Michigan State University, Entomology Department. Hormesis: Adaptive responses in biology and medicine. September 18, 2014.

Calabrese EJ. Hormesis and homeopathy. Lecture 3. Hormetic mechanisms. 4th Australian Conference of Bioregulatory Medicine, Adelaide, Australia. September 12-15, 2014.

Calabrese EJ. Hormesis and homeopathy. Lecture 4. Hormetic applications. 4th Australian Conference of Bioregulatory Medicine, Adelaide, Australia. September 12-15, 2014.

Calabrese EJ. Thematic Session 3: Hormesis: Adaptive Response in Biology and Medicine. NAALT/WALT Joint Session Conference, Arlington VA. September 12, 2014.

Calabrese EJ. Enhancing Biological Performance: Occurrence, Mechanisms and Applications. Human Performance Program Review, Basic Research Innovation Collaboration Center, Arlington VA. September 11, 2014.

Calabrese EJ. Hormesis: Its scientific foundations and biochemical regulatory applications. Physicians for Civil Defense, Oregon Institute of Science and Medicine, Knoxville, TN. July 26, 2014.

Calabrese EJ. LNT theory: How the NAS misled the world on cancer risk assessment. Physcians for Civil Defense, Oregon Institute of Science and Medicine, Knoxville, TN. July 26, 2014.

Calabrese EJ. Optimizing pre- and post-conditioning clinical outcomes: A dose response perspective. 13th Annual International Conference on Dose-Response. Preconditioning Adaptive Responses in Biology and Medicine. Building Biological Shields Against Disease and Injury. April 22, 2014.

Shamoun DY, Calabrese EJ. Risk assessment report card. 13th Annual International Conference on Dose-Response. Preconditioning Adaptive Responses in Biology and Medicine. Building Biological Shields Against Disease and Injury. April 22, 2014.

Shamoun DY, Calabrese EJ. Model uncertainty in cancer risk assessment. 13th Annual International Conference on Dose-Response. Preconditioning Adaptive Responses in Biology and Medicine. Building Biological Shields Against Disease and Injury. April 22, 2014.

Calabrese EJ. Hormesis: A looming scientific revolution in environmental regulation? Clark University, Worcester, MA. April 10, 2014.

Calabrese EJ. Hormesis and homeopathy. Lecture 1. Hormesis: biological foundations. Brauer Professional Conference, Adelaid, Australia. March, 2014.

Calabrese EJ. Hormesis and homeopathy. Lecture 2. Historical foundations of hormesis. Brauer Professional Conference, Adelaide, Australia. March, 2014.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. Office of Food Additives, CFSAN, FDA, College Park MD. March 19, 2014.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. Bridgewater State University, Bridgewater MA. February 21, 2014.

Calabrese EJ. Problems in the analysis used in launching the linear extrapolation approach for cancer risk assessment. American Chemical Council. February 20, 2014.

Calabrese EJ. Hormesis: Adaptive responses in biology and medicine. Duquesne University, Pittsburgh PA. February 13, 2014.

Calabrese EJ. How the US NAS misled the world community on cancer risk assessment. Bettis Atomic Power Lab, Pittsburgh, PA. February 12, 2014.

Calabrese EJ. Overthrowing the regulatory paradigm for carcinogens. Cato Institute, Capital Hill Briefing,Washington, DC. January 28, 2014

Calabrese EJ. Hormesis and the development of biological shields. TNO/Samueli Institute, The Netherlands. January 13, 2014.

2013

Calabrese EJ, Yazigi D. (2013). New methods in cancer risk assessment. Society of Risk Analysis, Baltimore MD. December 10, 2013.

Calabrese EJ. (2013). Comment at the Convocation for the Faculties of Engineering, Health Sciences, and Science of an honorary doctrate. Hormesis: How i got started. McMaster University, Hamilton, ON. November 22, 2013.

Calabrese EJ. (2013). Origins of the LNT: Department of Radiological Science, McMaster University, Hamilton ON. November 21, 2013.

Calabrese EJ. (2013). Hormesis: A basic biological concept. University of Michigan. November 15, 2013.

Calabrese EJ. (2013). Hormesis: Its role in toxicology and radiological health. University of Michigan. November 15, 2013.

Calabrese EJ. (2013). How the US NAS misled the world community on cancer risk assessment. MI American Nuclear Society. Ann Arbor, MI. November 14, 2013.

Calabrese EJ. (2013). Hormesis: Toxicological foundations, mechanisms and biomedical/clinical applications. Air Force Office of Scientific Research Human Performance and Biosystems Program. Basic Research Innovation Collaboration Center. Arlington, VA. October 30, 2013.

Calabrese EJ. (2013). Soil ingestion rates in children and adults: Implications for human health risk assessment. International Conference on Soils, Sediments, Water and Energy. University of Massachusetts, Amherst, MA. October 22, 2013.

Calabrese EJ. (2013). Soil, sediment, and dust ingestion pathway in human health and ecological risk assessment. International Conference on Soils, Sediments, Water and Energy. University of Masachusetts, Amherst, MA. October 22, 2013.

Calabrese EJ. (2013). Hormesis: Its toxicological foundations and therapeutic implications. School of Pharmacy, University of Connecticut, Storrs, CT. October 9, 2013.

Calabrese EJ. (2013). Low dose radiation therapy induces and anti-inflammatory phenotype: Biomedical implications. Environmental Mutagen Society Inflammation Symposium. September 23, 2013.

Calabrese EJ. (2013). Evolution of the linear no threshold model of radiation injury. American Association of Physicists in Medicine, 54th Annual Meeting. Indianapolis. August 5, 2013.

Calabrese EJ. (2013). Hormesis: Its biomedical foundations and therapeutic implications. American Association of Naturopathic Physicans. Keystone, Colorado. July 10-13, 2013.

Calabrese EJ. (2013). Hormesis theory. The revolution of diet: stay hungry, stay healthy. SBS TV Network (S. Korea). June 14, 2013.

Calabrese EJ. (2013). A method to evaluate hormesis in nanoparticle dose-response. 5th International Symposium - Nutrition, Oxygen Biology and Medicine. Paris France. June 5-7, 2013.

Calabrese EJ. (2013). Does it or doesn't it? Evidence for the existence of non-monotonic dose resonse. Webinar. Society of Toxicology Risk Assessment Specialty Section. San Antonio, TX. May 8, 2013.

Calabrese EJ. (2013). Origin of the linearity-no threshold (LNT) dose response concept. Dose-Response 2013: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst. April 23, 2013.

Calabrese EJ, Calabrese V. (2013). Low dose radiation therapy (LD-RT) is effective in the treatment of arthritis: Animal model findings. Dose-Response 2013: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst. April 23, 2013.

Calabrese EJ, Dhawan G. (2013). The historical use of radiotherapy in the treatment of sinus infections. Dose-Response 2013: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst. April 23, 2013

Calabrese EJ. (2013). History of the dose response. Web interview and presentation. Atoms for Peace, Italy. April 11, 2013

Calabrese EJ. (2013). Hormesis: Scientific revolution in environmental regulations. Department

of International Development, Community and Environment. Clark University, Worcester, MA. April 2, 2013.

Calabrese EJ. (2013). A looming scientific rebolutionin environmental regulations? Cato Institute. Washington DC. March 21, 2013.

Calabrese EJ. (2013). Hormesis. Research Training Group Annual Meeting. Jena, Germany. January, 2013.

2012

Calabrese, E.J. (2012). Chemical and radiation hormesis: Toxicological foundations and biomedical applications. Uniformed Services University, Armed Forces Radiobiology Research Institute. Bethesda, MD. November 30, 2012.

Calabrese, E.J. (2012). Hormesis: Toxicological and Risk Assessment Implications. University of Massachusetts, School of Public Health and Health Sciences. Presented to medical students and faculty from Russia, Novgorod State University. Amherst, MA. October 26, 2012.

Calabrese, E.J. (2012). The hormesis dose response. University of Louisville, Louisville. KY. October 24, 2012.

Calabrese, E.J. (2012). Hormesis: Its biological foundations and therapeutic implications. European Society of Integrative Medicine. Florence, Italy. September 20-21, 2012.

Calabrese, E.J. (2012). Hormesis: Its significance for toxicology, risk assessment and medicine. Plymouth Marine Laboratory. Plymouth, UK. July 18, 2012.

Calabrese, E.J. (2012). The hormetic dose response. United Kingdom Environmental Mutagen Society, Taliesin Arts Centre, Swansea Univeristy. Swansea Wales. July 15-18,2012.

Calabrese, E.J. (2012). How the LNT myth was launched. American Nuclar Society Annual Meeting. Hyatt Regency Hotel. Chicago, IL. June 25, 2012.

Calabrese, E.J. (2012). The hometic dose response. European Food Safety Authority Scientific Colloquium XVII on Low Dose Response in Toxicology and Risk Assessment. Parma, Italy. June 14, 2012 (video presentation).

Calabrese, E.J. (2012). Muller's deceptive Nobel Prize lecture and its risk assessment implications. New England Health Physics Society Symposium, Westford, MA. May 24, 2012.

Golden, R., and Calabrese, E.J. (2012). Re-evaluation of the LNT. Society of Toxicology Annual Meeting, San Francisco, CA. March 11-15, 2012.

Calabrese, E.J. (2012). Hormesis and the Salk polio vaccine. International Conference on Dose-Response 2012, University of Massachusetts. Amherst, MA. April 24-25, 2012.

Calabrese, E.J. (2012). Key historical studies serving as the basis for the linear dose response challenged. International Conference on Dose-Response 2012, University of Massachusetts. Amherst, MA. April 24-25, 2012.

Calabrese, E.J., and Dhawan, G. (2012). The role of x-rays in the treatment of gas gangrene: A historical assessment. International Conference on Dose-Response 2012, University of Massachusetts. Amherst, MA. April 24-25, 2012.

Golden, R., and Calabrese, E.J. (2012). Revisiting assumptions of linearity for radiation-induced cancer: Implications for chemical cancer risk assessment. International Conference on Dose-Response 2012, University of Massachusetts. Amherst, MA. April 24-25, 2012.

Sarill, M.A., and Calabrese, E.J. (2012). Biphasic dose responses to phytoestrogens: An evaluation of mechanisms. International Conference on Dose-Response 2012, University of Massachusetts. Amherst, MA. April 24-25, 2012.

Calabrese, E.J. (2012). Hormesis: Its significant for toxicology, pharmacology and drug development. Tufts University, Medford/Somerville, MA. April 17, 2012.

Calabrese, E.J. (2012). The hormetic dose response. Hormesis Research Training Group at the Friedrich-Schiller-University, Jena, Germany. Opening Ceremonies February 14, 2012.

Calabrese, E.J. (2012). Hormesis: Its significance for toxicology, pharmacology and drug development. Computer Science Department, University of Massachusetts. Amherst, MA. April 10, 2012.

Calabrese, E.J. (2012). School of Marine Sciences, University of Massachusetts, Amherst, MA. February 1, 2012.

Calabrese, E.J. (2012). Hormesis: a dose response revolution. IMMAG, Georgia Health Sciences University. Augusta, GA, January 23, 2012.

2011

Calabrese, E.J. (2011). Hormesis: Its significance for toxicology, pharmacology, and risk assessment. 43rd Society of Toxicology of Canada. Montreal, Canada, December 4-6, 2011.

Calabrese, E.J. (2011). When science fails society: Toxicology's 20th century legacy. Joint Meeting of the New England Sections of American Physical Society, American Association of Physics Teachers and the Soceity of Physics Student, Physics Department, University of Massachusetts, Amherst MA. November 19, 2011.

Calabrese, E.J. (2011). How toxicology got the dose response half right. FISH/BSU seminar. Bridgewater State University, Bridgewater, MA. November 18, 2011.

Calabrese, E.J. (2011). Hormesis: enhancing biological performance. Department of the Air Force – Photo-Electric-Magnetic-Bio-Stimulation (PEMB) Workshop. San Antonio, TX. October 31-November 1, 2011.

Calabrese, E.J. (2011). Harvard School of Public Health, Harvard University, JBL Symposium. Boston, MA. October 29, 2011.

Calabrese, E.J. (2011). Hormesis: Its significance for risk assessment and regulatory agencies. Mary Kay O'Connor Process Safety Center. Texas A&M University, College Station, Texas. October 25-26, 2011.

Calabrese, E.J. (2011). Hormesis: Its significance for toxicology, pharmacology and risk assessment. University of Connecticut, Advanced Toxicology Seminar. Connecticut. October 12, 2011

Calabrese, E.J. (2011). Hormesis: Its significance for food safety. Food Safety versus Food Secutiry – A Global Challenge. Wageningen, The Netherlands. October 4, 2011.

Calabrese, E.J. (2011). U-Shaped dose response curves. RIKILT, Wageningen, The Netherlands. October 3, 2011.

Calabrese, E.J. (2011). Hormesis: Its significance for toxicology, pharmacology and risk assessment. Prevention and Intervention: From Molecular Biology to Clinical Perspectives, Halle, Germany. September 16-18, 2011.

Calabrese, E.J. (2011). Hormesis; Its significance for toxicology, pharmacology and drug development. FDA Center for Drug Evaluation and Research (CDER), Rockville, MD. September 12, 2011.

Calabrese, E.J. (2011). Hormesis: Its significance for toxicology, pharmacology and risk assessment. Colloque ARET, Museaum National d'Histoire Naturelle, Paris, France. June 20-21, 2011.

Calabrese, E.J. (2011). Hormesis: Its significance for toxicology, pharmacology and riak assessment. Mary Kay O'Connor Process Safety Center, Texas A & M University, College Station, TX. May 5, 2011.

Nascarella, M.A., and Calabrese, E.J. (2011). Characterization of the biphasic antioxidant response of human cells to multi-walled carbon nanotubes. The 10th Annual International Conference on Dose-Response 2011: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst, MA. April 26-27, 2011.

Calabrese, E.J., and Stanek III, E.J. (2011). Hormesis demonstrated for mutagenicity. The 10th Annual International Conference on Dose-Response 2011: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst, MA. April 26, 2011.

Nascarella, M.A., and Calabrese, E.J. (2011). Case study: Quantitative assessment of the biphasic dose-response of polyN-isoproplacrylamide (PNIPAM) nanoparticles. The 10th Annual International Conference on Dose-Response 2011: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst, MA. April 26, 2011.

Stanek III, E.J., and Calabrese, E.J. (2011). Simulation studies to complement observational data: what can we learn? How should they be used? The 10th Annual International Conference on Dose-Response 2011: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst, MA. April 27, 2011.

Calabrese, E.J. (2011). Hormesis: Changing how we think about toxicology, medicine and risk assessment. Endocrine Disruptive Effects of Pesticides from Low Dose Exposure: Evidence for Non-Monotonic Dose Response Curves? The SAFE Consortium. Brussels, Belgium, March 12-17.

Calabrese, E.J. (2011). When Science Fails Society: Toxicology's 20th Century Legacy. Howard University, Washington, DC. March 30, 2011.

2010

Calabrese, E.J. (2010). Hormesis: Its scientific foundations and biomedical implications. L'Oreal, Clichy, France. November 3, 2010.

Calabrese, E.J. (2010). Hormesis: A revolution in toxicology, medicine, and risk assessment. Clark University, November 18, 2010.

Calabrese, E.J. (2010). 6th International Workshop on the CCN Family of Genes. International CCN Society. Belfast, Northern Ireland. October 20, 2010.

Calabrese, E.J. (2010). Hormesis: Its scientific foundations and biomedical implications. State University of New York, Albany, New York. October 8, 2010.

Calabrese, E.J. (2010). Hormesis: A revolution in toxicology, medicine, and risk assessment. Skidmore College, Skidmore, New York. October 7, 2010.

Calabrese, E.J. (2010). Historical blunders: How EPA got the dose response half right. Iona College, New Rochells, New York. September 30, 2010.

Calabrese, E.J. (2010). Historical blunders: The road to linearity. McMaster Univeristy, International Scientific Symposium, Ontario, Canada. August 26, 2010.

Calabrese, E.J. (2010). Hormesis: Scientific foundations and public health implications. Fermented Beverages and Health: Enhancement of Biological Responses Relevant for Human Health. Madrid, Spain. July 14, 2010.

Calabrese, E.J. (2010). Hormesis applications for neurodegenerative diseases. Drug Development for Neurodegenerative Diseases, Boston, MA. May 18, 2010.

Calabrese, E.J., Baldwin, L.A., and Leonard, D.A. (2010). The history of chemical hormesis. Presented at the 9th Annual International Conference: Dose-Response: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts-Amherst, MA. April 27-28, 2010.

Calabrese, E.J., Baldwin, L.A., and Leonard, D.A. (2010). The history of radiation hormesis. Presented at the 9th Annual International Conference: Dose-Response: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts-Amherst, MA. April 27-28, 2010.

Calabrese, E.J. (2010). Hormesis Update 2010. Presented at the 9th Annual International Conference: Dose-Response: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts-Amherst, MA. April 27-28, 2010.

Calabrese, E.J., and Nascarella, M.A. (2010). The frequency of hormetic responses in the Ames Assay. Presented at the 9th Annual International Conference: Dose-Response: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts-Amherst, MA. April 27-28, 2010.

Mosakowski, T., and Calabrese, E.J. (2010). Hormesis research in the People's Republic of China: Past trends in the academic literature and future directions. Presented at the 9th Annual International Conference: Dose-Response: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts-Amherst, MA. April 27-28, 2010.

Iavicoli, I., Calabrese, E.J., and Nascarella, M.A. (2010). Exposure to nanoparticles and hormesis. Presented at the 9th Annual International Conference: Dose-Response: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts-Amherst, MA. April 27-28, 2010.

Nascarella, M.A., and Calabrese, E.J. (2010). A Case Study: the risk of a hormetic response chemotherapy treatment. Society of Risk Analysis – New England Meeting. Camp, Dresser, Mckee, Cambridge, MA. April 1, 2010.

Calabrese, E.J. (2010). Hormesis in Toxicology and Pharmacology. Maastricht University. The Netherlands, March 26, 2010.

Calabrese, E.J. (2010). The limits of legislation. Koopman International European Commission and Parliament and joint Centre for European Policy Studies. Brussels, March 25, 2010.

Calabrese, E.J., and Nascarella, M.A. (2010). Estimating the frequency of hormesis in the Ames assay. Presented at the Society of Toxicology Annual Meeting, Salt Lake City, UT. March 10, 2010.

Calabrese, E.J. (2010). Estimating the frequency of hormesis in the Ames Assay. To be presented at the 49th Annual Meeting of the Society of Toxicology. Salt Lake City, UT. March 7-11, 2010.

Calabrese, E.J. (2010). Hormesis: Why it Transforms Toxicology, Molecular Biology and Clinical Medicine. Brown University, Providence, RI. March 3, 2010.

Calabrese, E.J. (2010). Hormesis: Why it transforms toxicology and risk assessment. Presented at Worcester Polytechnic Institute-REACH. February 5, 2010.

Calabrese, E.J. (2010). Hormesis is central to pharmacology and toxicology. Northeastern University, Boston MA. January 21, 2010.

2009

Calabrese, E.J. (2009). Hormesis: State of the science. Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD. December 9, 2009.

Calabrese, E.J., and Nascarella, M.A. (2009). Hormesis: Scientific foundations and risk assessment implications. Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD. December 9, 2009.

Lewis, S.C., and Calabrese, E.J. (2009). Hormesis: Barriers for regulatory risk assessment. Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD. December 6-9, 2009.

Jones, A.C., Anderton, D.L., Stanek, E.J., and Calabrese, E.J. (2009). Survey Results for the hormesis knowledge and opinion survey administered to risk assessment and toxicology professionals. Presented at the Society for Risk Analysis Annual Meeting, Baltimore, MD. December 6-9, 2009.

Calabrese,E.J. (2009). Hormesis Enhances Environmental Toxicology Research and its Applications. Presented at William & Mary, Virginia Institute of Marine Science. Gloucester Point, VA. November 6, 2009.

Calabrese, E.J. (2009). Hormesis: Why it should transform toxicology and pharmacology. Northeast Chapter of the Society of Toxicology, Cambridge, MA. October 16, 2009.

Calabrese, E.J. (2009). Hormesis is central to biology and medicine. University of Rhode Island, Kingston, RI. October 13, 2009.

Calabrese, E.J. (2009). Hormesis is central to biology and medicine. 8th LOWRAD International Conference, Rio de Janeiro, Brazil. September 28-30, 2009.

Calabrese, E.J. (2009). Hormesis: Scientific foundations and risk assessment implications. ExxonMobil Biomedical Sciences, Inc., Iselin, NJ. September 15, 2009.

Calabrese, E.J. (2009). Hormesis: A central concept in biology and carcinogenesis. The University of Vermont, Department of Pathology, Burlington, VT. September 14, 2009.

Calabrese, E.J. (2009). Hormesis and Medicine. Boiron. Lyon, France. June 22-23, 2009

Nascarella, M., Beck, B., and Calabrese, E.J. (2009). Quantifying Hormetic (Biphasic) Dose-Responses in the Assessment of Nanoparticle Toxicology. International Conference on the Environmental Implications and Applications of Nanotechnology, June 9-11, 2009

Calabrese, E.J. (2009). Hormesis: A dose response revolution. New England Chapter of the Health Physics Society annual meeting, Westford, MA. June 4, 2009.

Nascarella, M.A., and Calabrese, E.J. (2009). A Comparison of Multiple Methods to Evaluate Biphasic (Hormetic) Dose Responses in High-Throughput In Vitro Toxicology Screens. NRC Symposium on Toxicity Pathway-Based Risk Assessment: Preparing for Paradigm Change. May 11-13, 2009.

Calabrese, E.J. (2009). Hormesis: A dose response revolution. Binghamton University, State University of New York. April 17, 2009.

Calabrese, E.J. (2009). Challenging the assumptions about toxicological dose response: Scientific, ethical and policy implications of hormesis. Clark University. March 20, 2009.

Stanek, E.J. III and Calabrese, E.J. (2009). Meta Analysis of Soil Ingestion Intake for Childhood Risk Assessment, Eastern North American Region Biometrics Meetings, March 16, 2009, San Antonio, Texas.

Nascarella, M.A., and Calabrese, E.J. (2009). The relationship between IC50, toxic threshold, and the magnitude of stimulatory response in biphasic (hormetic) dose-responses. Society of Toxicology Annual Meeting, Baltimore, MD. March 15-19, 2009.

Jones, A.C., Anderton, D.L., Stanek, E.J., and Calabrese, E.J. (2009). Hormesis knowledge and opinion survey results. Presented at the Society of Toxicology Annual Meeting, Baltimore, MD. March 15-19, 2009.

Calabrese, E.J. (2009). Hormesis: What it means for toxicology, the environment and public health. FISH Spring 2009, Biology Department Seminar Hour. Bridgewater State University. Februaru 27, 2009.

Calabrese, E.J. (2009). Hormesis: What it means for toxicology, the environment and public health. Plant and Soil Science, University of Massachusetts, Amherst, MA. February 3, 2009.

2008

Stanek, E.J. III and Calabrese, E.J. (2008). Exposure Assessment for Children: Soil Ingestion, Indian Statistical Institute Seminar, Oct 31, 2008, ISI Kolkata, India.

Nascarella, M.A., and Calabrese, E.J. (2008). Characterizing the quantitative features of hormetic dose-responses in a single high-throughput assay evaluating anticancer agents. To be presented at the Society for Risk Analysis Annual Meeting, Boston, MA. December 8, 2008.

Stanek, E., and Calabrese, E.J. (2008). Exposure assessment in children: Soil ingestion. Indian Statistical Institute, Kolkata, India. October 31, 2008.

Jones, A.C., Anderton, D.L., Stanek, E.J., and Calabrese, E.J. (2008). Hormesis knowledge and opinion survey results. Presented at the Society of Toxicology Northeast Regional Chapter Fall Meeting, Shrewsbury, MA. October 24, 2008.

Nascarella, M.A., and Calabrese, E.J. (2008). Toxic potency and hormesis in dose-response assessment. Presented at the Society of Toxicology Northeast Regional Chapter Fall Meeting, Shrewsbury, MA. October 24, 2008.

Calabrese, E.J. (2008). Hormesis: a central concept in biology, the biomedical sciences and toxicology. University of Connecticut, Storrs, CT. October 22, 2008.

Waters, D.J., and Calabrese, E.J. (2008). The U-shaped curve: when more is not better. Environmental Mutagen Society. Puerto Rico. October 21, 2008.

Calabrese, E.J. (2008). Hormesis: What it means for pharmacology and toxicology. The Boston Area Pharmaceutical Toxicology Group (BAPTG). Novartis Institutes of Biomedical Research, Inc., Cambridge, MA. September 18, 2008.

Calabrese, E.J. (2008). Why I think hormesis is the most fundamental dose response relationship in biology. McMasters University, Hamilton, Canada. September 15, 2008.

Calabrese, E.J. (2008). Hormesis: Its significant to toxicology, risk assessment and Medicine. North American Congress of Clinical Toxicology, Toronto, Canada. September 14, 2008.

Calabrese, E.J. (2008). Hormesis and the pharmaceutical industry. Millennium Pharmaceuticals, Inc, Cambridge, MA. June 16, 2008.

Calabrese, E.J. (2008). Separating stimulant and impairing function in hormetic profiles with independent component analysis (ICA). The 7th Annual International Conference – Session II: Biomedical. Dose-Response 2008: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst, MA. April 29, 2008.

Calabrese, E.J. (2008). Hormesis – 2008 – Current Status. The 7th Annual International Conference – Session I: Plenary. Dose-Response 2008: Implications for Toxicology, Medicine, and Risk Assessment. University of Massachusetts, Amherst, MA. April 29, 2008.

Nascarella, M.A., Stanek, E.J., and Calabrese, E.J. (2008). Evaluating stimulatory cell proliferation in anticancter drug dose-responses. University of Massachusetts School of Public Health and Health Sciences, 11th Annual Poster Session. University of Massachusetts, Amherst, MA. March 27, 2008

Nascarella, M.A., Stanek, E.J., and Calabrese, E.J. (2008). The quantitative evaluation of hormesis in anticancer drug dose-response. Society of Toxicology Annual Meeting. Seattle, WA. March 19, 2008.

Calabrese, E.J. (2008). Hormesis: The most fundamental dose response model. University of Ottawa Seminar, Ottawa, Canada. February 1, 2008.

Calabrese, E.J. (2008). Hormesis: Improving health reducing costs. Health Canada Seminar, Ottawa, Canada. January 31, 2008.

2007

Nascarella, M.A., and Calabrese, E.J. (2007). The quantitative characterization of the dose-response relationship of a panel of yeast (*Saccharomyces cerevisiae*) strains to prospective anticancer agents. Society for Risk Analysis Annual Meeting. San Antonio, TX. December 12, 2007.

Calabrese, E.J. (2007). Hormesis: The most fundamental dose response model. Department of Kinesiology, University of Massachusetts, Amherst, MA. October 29, 2007.

Nascarella, M.A., Stanek, E.J., and Calabrese, E.J. (2007). The quantitative characterization of hormesis in the National Cancer Institute's Yeast Anticancer Drug Screen Data. Society of Toxicology Northeast Regional Chapter, Fall Meeting. Groton, CT. October 26, 2007.

Calabrese, E.J. (2007). Hormesis and its relevance for clinical psychology. Harvard Medical School, Newton, MA. March 12, 2007.

2006

Calabrese, E.J. (2006). Hormesis: How it may affect toxicology and pharmacology. Sanofi-Aventis U.S. Inc. Bridgewater, MA. November 15, 2006.

Calabrese, E.J., and Stanek, E.J. (2006). Arsenic bioavailability in humans. The Gradient Corporation. Cambridge, MA. October 5, 2006.

Calabrese, E.J. (2006). Historical foundations of hormesis. University of Kansas Medical Center, Kansas City, KS. October 3, 2006.

Calabrese, E.J. (2006). Hormesis scientific foundations. University of Kansas Medical Center, Kansas City, KS. October 3, 2006.

Calabrese, E.J. (2006). Hormesis as a vehicle for therapeutic agents. Therapeutic Discovery Conference. Rensillierville, NY. September 10, 2006.

Staudenmayer, J. and Calabrese, E.J. (2006). Hormesis is more common than the threshold model in large NCI yeast database study. International Hormesis Society Conference. University of Massachusetts, Amherst, MA. June 7, 2006.

Calabrese, E.J. (2006). Hormesis: scientific foundations. Florence, Italy. April 7, 2006.

Calabrese, E.J. (2006). Hormesis: A challenge to the linear dose-response model, and its implications in risk assessment, regulatory policy, and biomedical research. Society of Toxicology 2006 45th Annual Meeting & ToxExpo. San Diego, CA. March 8, 2006.

Calabrese, E.J. (2006). Hormesis: Toxicological update and potential applications to the air force. Air Force Office of Scientific Research, Arlington, VA. January 31, 2006.

Calabrese, E.J. (2006). Experimental data relevant to single dose cancer assessment. 31st Annual Winter Toxicology Forum, Washington, DC. January 31, 2006.

Calabrese, E.J. (2006). Hormesis: Scientific development and implications for risk assessment. 31st Annual Winter Toxicology Forum, Washington, DC. January 31, 2006.

Calabrese, E.J. (2006). Hormesis: Common, generalizable and significant. Eli Lilly Research Laboratories, Greenfield, IN. January 11, 2006.

2005

Calabrese, E.J. (2005). Hormones is Important for Toxicologists and Risk Assessors: The Case for Hormesis as the Most Fundamental Dose Response Relations. Michigan State University, MI. December 8, 2005.

Calabrese, E.J. (2005). Historical Foundations of the Dose Response. Michigan State University, MI. December 8, 2005.

Calabrese, E.J. (2005). Soil Ingestion in Children and Adults. RIVM, Utrecht, The Netherlands. November 7, 2005.

Calabrese, E.J. (2005). Introduction of the Concept of Hormesis: Implications for Risk Assessment.. Utrecht University, Utrecht, The Netherlands. November 8, 2005.

Calabrese, E.J. (2005). Hormesis: Historical Perspectives, and Recent Advances. Health Council of the Netherlands, The Hague, The Netherlands. November 9, 2005.

Calabrese, E.J. (2005). Hormesis: Societal Implications. International Policy Network & the Institute of Economic Affairs. London, United Kingdom. November 10, 2005.

Calabrese, E.J. (2005). Hormesis and Its Impact on Future Toxicity Testing Strategies. National Research Council, Committee on Toxicity Testing and Assessment of Environmental Agents, Washington, DC. October 20-21, 2005.

Calabrese, E.J. (2005). Hormesis Its Impact on Toxicology and Risk Assessment. Yale University, New Haven, CT. October 6, 2005.

Calabrese, E.J. (2005). Is There Non-Random Biological Activity Below the NOAEL? Center for Risk Science and Communications. University of Michigan, Ann Arbor, MI. September 16, 2005.

Calabrese, E.J. (2005). Hormesis: Challenging the EPA Dose Response Paradigm. Environmental Management Association. Annual Sound Science Seminar, Michigan. September 15, 2005.

Calabrese, E.J. (2005). The Emergence of Hormesis in Biology, Toxicology and Medicine. 4th International BELLE Conference, University of Massachusetts, Amherst, MA. June 6, 2005.

Calabrese, E.J. (2005). Scientific underpinnings of hormesis. European Union. Video Presentation. Italy, May 19, 2005.

Calabrese, E.J. (2005). Costing the Earth. BBC Radio interview. April 5, 2005.

Calabrese, E.J. (2005). Biomedical and Clinical Implications of Hormesis (Guest Speaker). Annual Meeting Franklin and Hampshire Districts of Massachusetts Medical Society. Sunderland, MA. April 20, 2005

Calabrese, E.J. (2005). Hormesis Seminar. US EPA. Research Triangle Park, NC. April 26-27, 2005.

Calabrese, E.J. (2005). Soil Ingestion Estimation in Children and Adults: A Dominant Influence in Site-Specific Assessment. Health Canada Environmental and Occupational Toxicology Seminar Series. March 23, 2005.

Calabrese, E.J. (2005). Hormesis: The Dose-Response Revolution. Health Canada Environmental and Occupational Toxicology Seminar Series. March 24, 2005.

2004

Calabrese, E.J. (2004). Hormesis as a biological concept. Amherst College. October 25, 2004.

Calabrese, E.J. (2004). Hormesis as a concept in Toxicology. Holy Cross College. Worcester, MA. October 20, 2004.

Calabrese, E.J. (2004). Hormesis Roundtable. American Industrial Hygiene Conference and Exposition. Atlanta, GA. May 13, 2004.

Calabrese, E.J. (2004). Hormesis and the LNT. MIT. Cambridge, MA. April 1, 2004.

Stanek III, E.J., and Calabrese, E.J. (2004). Arsenic bioavailability in humans. Environmental Institute, University of Massachusetts. March 26, 2004.

Blain, R.R., and Calabrese, E.J. (2004). Hormesis database. Society of Toxicology 43rd Annual Meeting. Baltimore, MD. March 24, 2004.

Calabrese, E.J. (2004). Hormesis: Its implications for hazard and risk assessment. Society of Toxicology 43rd Annual Meeting. Baltimore, MD. March 22, 2004.

Ewald, K.A., and Calabrese, E.J. (2004). Protection against mechanistically distinct hepatotoxicants is associated with acute phase response. Society of Toxicology 43rd Annual

Meeting. Baltimore, MD. March 22, 2004.

Calabrese, E.J. (2004). Hormesis and its implications for State health departments (Video Conference). Marin County Health Department. California. February 27, 2004.

Calabrese, E.J. (2004). Hormesis and its implications for aging. National Institute of Aging. Baltimore, MD. February 26, 2004.

Calabrese, E.J. (2004). Hormesis and new developments in assessing the dose-response. Johns Hopkins University. Baltimore, MD. February 25, 2004.

Calabrese, E.J. (2004). Hormesis and public health. Environmental Media Services. Washington, DC. February 25, 2004.

2003

Nascarella, M.A., and Calabrese, E.J. (2003). Stage specific toxicity and the hormetic dose response relationship in the black blowfly. 3rd Annual Institute of Environmental and Human Health Toxicology Exposition. Lubbock, TX. April 4, 2003.

Calabrese, E.J. (2003). Toxicological Foundations of Hormesis. Canadian Society of Toxicology. Plenary Address. Montreal, Canada. December 2003.

Calabrese, E.J. (2003). Hormesis and its role in Toxicology. Society of Toxicology of Canada. Plenary Session. Montreal, Canada. December 8, 2003.

Calabrese, E.J. (2003). The dose-response relationship: A new paradigm with broad biomedical implications. University of Massachusetts School of Public Health. Amherst, MA. November 25, 2003.

Calabrese, E.J. (2003). Hormesis. Hazard and risk assessment. Texas Tech University (Video Conference). November 7, 2003.

Calabrese, E.J. (2003). Hormesis. College of the Holy Cross. Worcester, MA. October 14, 2003.

Calabrese, E.J. (2003). Hormesis and risk assessment. The Dow Foundation. Midland, MI. September 17, 2003.

Calabrese, E.J., and Baldwin, L.A. (2003). Hormesis at the NTP. Second Non-Linearity Dose Response Relationships in Biology, Toxicology and Medicine Conference. Amherst, MA. June 8, 2003.

Calabrese, E.J. (2003). Biomedical implications of hormesis. Second Non-Linearity Dose Response Relationships in Biology, Toxicology and Medicine Conference. Amherst, MA. June 8, 2003.

Calabrese, E.J. (2003). Non-Linearity Dose-Response Relationships in Biology, Toxicology and Medicine, International Conference. Amherst, MA. May 28, 2003.

Calabrese, E.J. (2003). Bowdoin College, Department of Chemistry. Hormesis: New Concepts in our Understanding of the Dose Response. Brunswick, ME. May 2, 2003.

Calabrese, E.J. (2003). Tufts University, Department of Environmental Engineering. Hormesis: Occurrence and Mechanistic Foundations. Medford, MA. April 17, 2003.

Calabrese, E.J. (2003). Tufts University Medical School. Medical Implications of Hormesis. Boston, MA. April 17, 2003.

Calabrese, E.J. (2003). Society of Environmental Toxicology and Chemistry (SETAC) North Atlantic Chapter, Annual Meeting. Hormesis: Occurrence, Generalizability and Applications to Toxicology and Risk Assessment. Mystic, CT. April 24, 2003.

Calabrese, E.J. (2003). Columbia University, School of Education. Hormesis: Conceptual Framework and Application to Environmental Science Curriculum. New York. February 17, 2003.

Calabrese, E.J. (2003). Boston University School of Public Health. Biphasic Dose Response Relationships in Biology, Toxicology and Medicine. February 21, 2003.

Calabrese, E.J. (2003). Are Human Exposure Limits Too Conservative? Non-Linear Dose Response Relationships and "Hormesis". Aberdeen Proving Grounds. January 14, 2003.

2002

Nascarella, M.A., and Calabrese, E.J. (2002). A model system to explore the hormesis dose response relationship. Society for Risk Analysis Annual Meeting. New Orleans, LA. December 9, 2002.

Nascarella, M.A., Stoffolano, J.G., and Calabrese, E.J. (2002). Hormesis and stage specific toxicity induced by cadmium in an insect model, the queen blowfly, *Phormia regina* Meig. Society for Environmental Toxicology and Chemistry Annual North American Meeting. Salt Lake City, UT. November 19, 2002.

Calabrese, E.J. (2002). International Conference on Chemical Mixtures (ICCM). Atlanta, GA. September 11, 2002.

Calabrese, E.J. (2002). Toxicological Risk Assessment of DIMP. Colorado Water Quality Control Commission. Denver, CO. December 10, 2002.

Calabrese, E.J. (2002). Hormesis and High Risk Groups. UMDNJ – New Jersey Medical School. November 14, 2002.

Calabrese, E.J. (2002). The Hormetic or Threshold Model: Which is the Most Common Phenomenon in Toxicology. Cornell University. November 7, 2002.

Calabrese, E.J. (2002). Federal-State and Risk Analysis Committee (FSTRAC). (2002). U-Shaped Dose Responses in Toxicology and their Risk Assessment Implications. October 23, 2002.

Calabrese, E.J. (2002). ATSDR. The Hormetic Dose-Response Model is More Common Than the Threshold Model in Toxicology. Atlanta, GA. September 11, 2002.

Calabrese, E.J. (2002). NCAC-SOT. U-shaped Dose Responses Curves – What, Why and How?. May 16, 2002.

Calabrese, E.J. (2002). Toxicological Foundation of Hormesis. Bates College. February 14, 2002.

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Nascarella, M.A., Stoffolano, J.G., and Calabrese, E.J. (2001). Stage specific and hormetic effects induced by cadmium in the black blowfly. American Public Health Association 129th Annual Meeting. Atlanta, GA. October, 2001.

Calabrese, E.J. (2001). Hormesis: Current Status. Medical College of New York, Department of Animal Science. November 28, 2002. November , 14 2001, GE S

Calabrese, E.J. (2001). U-shaped dose responses in biology, toxicology and medicine: frequency, quantitative features and possible significance. Yale University. October 3, 2001.

Calabrese, E.J. (2001). The history of the dose-response relationship: reassessing the foundation of toxicology. Yale University. October 3, 2001.

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Calabrese, E.J. (2001). Harvard University. Hormesis. September 21, 2001.

Calabrese, E.J. (2001). Hormesis: Pharmacological and Toxicological Foundations. University of Rhode Island. June 31, 2001.

2000

Calabrese, E.J. (2000). Scientific data on low dose radiation and cancer. Health Benefits of Low Dose Radiation. Radiation, Science, and Health, Inc. Washington, DC. November 15, 2000.

Calabrese, E.J. (2000). Radiation Hormesis. BEIR VII Committee meeting. National Academy of Sciences, Washington, DC. September 20, 2000.

Calabrese, E.J. (2000). When adults are at greater risk than children. Conference on 10X factors. Hoffman-LaRoche, Nutley, NJ. May 3, 2000.

Calabrese, E.J. (2000). Hormesis as a biological phenomenon. Dept. of Entomology, University of Massachusetts, Amherst, MA. March 22.

Calabrese, E.J. (2000). The historical foundations of chemical hormesis. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J. (2000). The historical foundations of radiation hormesis. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

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Calabrese, E.J. (2000). Description of the chemical and radiation hormesis database. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

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Calabrese, E.J., and Baldwin, L.A. (2000). Apoptosis and biphasic response. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

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Calabrese, E.J., and Baldwin, L.A. (2000). Reproductive toxicity & U-shaped curves. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Adenosine: Biphasic receptor binding via allosteric enhancement. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Adenosine: Adenosine induces biphasic responses in renal vasculature. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Adenosine: Apomorphine induced biphasic penile erection: Occurrence and mechanistic basis. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic response of estrogens: Angiogenesis, bone formation, and immunostimulation. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic response of estrogens: Human breast cell proliferation, and DNA synthesis in human vascular cells. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic response of estrogens: Phytoestrogen and clot formation. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Cadmium induced biphasic responses. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of amyloid β-peptide. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic dose-response relationship between peripheral corticosterone and memory. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of nitric oxide: Osteoclast differentiation, macrophase synthesis of vitamin D3, and vasodilation in the human forearm. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of nitric oxide: Myocardial contraction, and calcium current in the heart. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of nitric oxide: Methylene blue and behavior, excitatory amino acids, and neutrophil migration. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of nitric oxide: Carbon monoxide induces a biphasic release of NO, biphasic effects of neuroleptic drugs on NOS, and NO and sperm function. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of testosterone: Chondrocytes, and sertoli cell function. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of testosterone: Prolactin, and prostate cancer cells (LNCaP). Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of prostaglandins: PGE_2 and verapamil, a calcium channel blocker, and bone formation. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of nonsteroidal anti-inflammatory drugs (NSAIDS): Prostaglandin production and transport. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of prostaglandins: neutrophil migration, corticosteroids, and transforming growth factor β. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

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Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of opiates: Cardiovascular and respiratory effects fetal breathing movements (FBM) in the lamb, neutrophil migration, peripheral blood lymphocyte (PBL) natural killer-activity, and corticosterone production. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of opiates: hCG secretion, HIV growth, and behavioral responses pain/euphoria. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of opiates: binding to brain receptors. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic chemotaxis effects of alcohol, alpha-1 proteinase inhibitor (API), FMLP, and mouse nerve growth factor. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic chemotaxis effects on neutrophils, tumor cells, and fibroblasts. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of dopamine: Background and biomedical significance, and prolactin secretion. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of dopamine: artery relaxation. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of dopamine: Apomorphine on pain, and locomotion. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Biphasic effects of dopamine agonists on memory. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

Calabrese, E.J., and Baldwin, L.A. (2000). Apoptosis and biphasic response. Poster presentation. Chemical and Radiation Hormesis Scientific Foundations. Amherst, MA. January 19-20.

1999

Calabrese, E.J. (1999). Toxicology of DIMP. National Academy of Sciences. Washington, DC. November 22.

Calabrese, E.J. (1999). Radiation hormesis: current status. U.S. Nuclear Regulatory Commission. Rockville, MD. March 29.

Calabrese, E.J. (1999). Hormesis and adaptation. NIH. Bethesda, MD. April 26.

Calabrese, E.J. (1999). Single exposure carcinogen database. U.S. EPA. Washington, DC. June 12.

Stanek, E.J., and Calabrese, E.J. (1999). Soil ingestion in children. U.S. EPA. Research Triangle Park, NC. June 14.

Calabrese, E.J. (1999). Health effects of DIMP. U.S. National Academy of Science. Washington, DC. November 4.

Calabrese, E.J. (1999). Radiation hormesis. NE Radiology Society. Boston, MA. June 18.

1998

Calabrese, E.J. (1998). New developments on hormesis. AIHC. Washington, DC. January 13.

Calabrese, E.J. (1998). Scientific foundations of hormesis. North Carolina Chapter of the Society of Risk Analysis. Carey, NC. April 27.

Calabrese, E.J. (1998). Chair – conference on societal implications of hormesis. Research Triangle Park, NC. October 5-6.

Calabrese, E.J. (1998). Historical foundations of chemical hormesis. Conference on societal implications of hormesis. Research Triangle Park, NC. October 5-6.

Calabrese, E.J. (1998). Implications of hormesis for risk assessment. 10X uncertainty factor conference. Medical College of New Jersey. Newark, NJ. November 11.

Calabrese, E.J. (1998). Non-monotonic dose-response relationships and their risk assessment implications. EPA National Symposium. Carey, NC. April 28.

Calabrese, E.J. (1998). Single exposure carcinogens and its implications for state public health risk assessors. National Teleconference Presentations for the ATHO Foundation. May 7.

Calabrese, E.J. (1998). U-shaped dose-response relationship. EPA Drinking Water Office-FASTAC. Boston, MA. May 8.

Calabrese, E.J. (1998). Hormesis and its risk assessment implications. Pfizer. Groton, CT. May 21.

Calabrese, E.J. (1998). Low dose responses to chemical stressor agents. General Electric. Schenectady, NY. July 30.

Calabrese, E.J. (1998). Hormesis and the biological effects of low level exposures. Occupational Safety and Health Group. Washington, DC. August 4-5.

1997

Calabrese, E.J. (1997). Dose-response relationships and endocrine disruption. Conference on Endocrine Disruption. Research Triangle Park, NC. January 13.

Calabrese, E.J. (1997). Single exposure carcinogen data base. NIOSH. Cincinnati, OH. January 23.

Calabrese, E.J., Blain, R.B., Leonard, D., and Ewald, K. (1997). Role of neutrophils and acute phase proteins in the hepatotoxic interaction between kepone and carbon tetrachloride. SOT Annual Meeting. Cincinnati, OH. March 10.

Calabrese, E.J., and Stanek, E.J. (1997). The amount of particle size of soil ingested by children. SOT Annual Meeting. Cincinnati, OH. March 12.

Calabrese, E.J., and Blain, R.B. (1997). Stress effects on carbon tetrachloride toxicity. SOT Annual Meeting. Cincinnati, OH. March.

Calabrese, E.J. (1997). Role of hormesis in risk management. DOD conference at NIH, Bethesda, MD. May 15.

Calabrese, E.J. (1997). Development of a chemical hormesis database: strengths, limitations, and generalized ability. Toxicology Forum. Aspen, CO. July 7-11.

Calabrese, E.J. (1997). Hormesis: Database and underlying mechanisms. Toxicology Forum, Aspen, CO. July 13.

Calabrese, E.J. (1997). Development of a chemical hormesis data base: Strengths, limitations, and generalized ability. Toxicology Forum. Aspen, CO. July 11.

Calabrese, E.J. (1997). Acute episodes of soil ingestion. Society of Risk Analysis. Washington, DC. December 7.

Calabrese, E.J. (1997). Single exposure carcinogens. Society of Risk Analysis. Washington, DC. December 7.

1996

Calabrese, E.J. (1996). Adaptive mechanisms and dose-response relationships. Texas A&M University. Texas. February, 19.

Calabrese, E.J. (1996). Acute exposures to genotoxic carcinogens. University of Montreal. Canada. April 10.

Calabrese, E.J. (1996). Current issues in risk assessment. Harvard University. Boston, MA. August 5.

Calabrese, E.J. (1996). Acute exposures to chemical carcinogens. National Academy of Sciences. Washington, DC. September 17.

Calabrese, E.J. (1996). Chemical hormesis. Texas Chemical Industry Association. Houston, TX. September 23.

Calabrese, E.J. (1996). Chaired session on genetic factors and environmental exposures. EPA Conference. Durham, NC. September 25.

Calabrese, E.J. (1996). Ecogenetics: genetic predisposition to toxic substances. EPA conference on interindividual differences in susceptibility. Durham, NC.

Calabrese, E.J. (1996). Single exposure carcinogens. New England Society for Occupational and Environmental Medicine. Boston, MA. December 5.

Calabrese, E.J. (1996). Chemical Hormesis. Safety factors in risk assessment. Nutley, NJ. December 6.

1995

Calabrese, E.J. (1995). Single exposure carcinogens. Society of Toxicology. Baltimore, MD. March 5-9.

Calabrese, E.J. (1995). Retrieval database on single exposure carcinogens. Society of Toxicology. Baltimore, MD. March 5-9.

Calabrese, E.J. (1995). Development of annual soil ingestion distributional estimates of 64 children based on daily soil ingestion values. Society of Toxicology. Baltimore, MD. March 5-9.

Schmidt, C.W., Leonard, D.A., Baldwin, L.A., Zhao, X.Q., and Calabrese, E.J. (1995). Administration of G2 activating agents modulates carbon tetrachloride induced hepatotoxicity. Society of Toxicology. Baltimore, MD. March 5-9.

Johnson, R.B., and Calabrese, E.J. (1995). The effects of repeat dosing and repeat blood withdrawal on carbon tetrachloride toxicity. Society of Toxicology. Baltimore, MD. March 5-9.

Calabrese, E.J. (1995). Uncertainty factors: their toxicological bases. Conference on Uncertainty Factors in Risk Assessment. NJ Medical School. Nutley, NJ. April 7.

Calabrese, E.J. (1995). Carcinogens that cause cancer with an single dose. Clark University. Worcester, MA. April 16.

Calabrese, E.J. (1995). Single exposure carcinogens. Risk Science Institute/Brookings Institute. l Washington, DC. April 23.

Calabrese, E.J. (1995). Soil ingestion in children and adults. Louisiana Department of Environmental Quality. Baton Rouge, LA. June 28.

Calabrese, E.J. (1995). Soil ingestion estimates. 10th Annual Soil Contamination Conference. University of Massachusetts. Amherst, MA. October 23.

Calabrese, E.J. (1995). Variability in response to toxic substances. Conference on Multiple Chemical Sensitivity. Baltimore, MD. October 30.

Calabrese, E.J. (1995). BELLE-An overview and a long-term view. Texas Chemical Industry

Institute. Houston, TX. November 7.

Calabrese, E.J. (1995). Tissue repair as a toxicological principle. American College of Toxicology. Vienna, VA. November 12.

1994

Calabrese, E.J. (1994). Biological effects of low level exposures to chemicals and radiation. Society of Risk Analysis. Baltimore, MD. December 7.

Calabrese, E.J. (1994). Chemical mixtures - a primer. US EPA. Raleigh, NC. November 7.

Calabrese, E.J. (1994). Current soil ingestion estimates. Louisiana State University. New Orleans, LA. November 2.

Calabrese, E.J. (1994). Children and adult soil ingestion. 9th Conference on Hydrocarbon Contaminated Soils. Amherst, MA. October 20.

Calabrese, E.J. (1994). BELLE as a concept. American College of Toxicology. Williamsburg, VA. October 26.

Calabrese, E.J. (1994). How to derive daily estimates of soil ingestion. U.S. EPA Exposure Assessment Group. Washington, DC. June 1.

Calabrese, E.J. (1994). Recent developments in soil ingestion. International Association for Lead and Zinc Industries. Chapel Hill, NC. May 26.

Calabrese, E.J. (1994). Biological effects of low level exposures (BELLE). American Occupational Health Conference. Chicago, IL. April 21.

Calabrese, E.J. (1994). Discussion of the key risk assessment issues. Symposium on Synthetic Vitreous Fibers: Scientific and Public Policy Issues. ISRTP. Arlington, VA. March 2-3.

Calabrese, E.J., and Mehendale, H.M. (1994). Cellular repair processes-the role of tissue repair as an adaptive strategy: why low doses are often non-toxic and why high doses can be fatal. Society of Toxicology 33rd Annual Meeting. Dallas, TX. March 1994.

Calabrese, E.J. (1994). Discussion of the key public policy issues and discussion of issues identified and recommendations made. Symposium on Synthetic Vitreous Fibers: Scientific and Public Policy Issues. ISRTP. Arlington, VA. March 2-3.

Calabrese, E.J. (1994). Chair session on unusual dose-response curves and implications for risk assessment. Society of Toxicology. Dallas, TX. March 17.

Calabrese, E.J. (1994). Cellular repair processes--the role of tissue repair as an adaptive strategy: why low doses are often non-toxic and why high doses can be fatal. Society of Toxicology Annual Meeting, Poster Discussion Session: Unusual Dose-Response Relationships: Mechanisms and Implications for Risk Assessment. Dallas, TX. March 17.

Calabrese, E.J. (1994). Session Chair. Unusual shaped dose-response curves. Society of Toxicology Annual Meeting, Poster Discussion Session: Unusual Dose-Response Relationships: Mechanisms and Implications for Risk Assessment. Dallas, TX. March 17.

Calabrese, E.J. (1994). A single exposure to certain chemical carcinogens can cause cancer: documentation, limitations and implications for risk assessment. US EPA Environmental Criteria and Assessment Office Seminar. Cincinnati, OH. March 30.

Calabrese, E.J. (1994). Single exposure carcinogens. Massachusetts Attorney's General. Boston, MA. January 13.

1993

Calabrese, E.J. (1993). The use of in vitro studies in the pursuit of improved animal extrapolation. World Congress on Alternative and Animal Use in the Life Sciences. Baltimore, MD. November, 14-19.

Calabrese, E.J. (1993). Soil ingestion estimates. ERM Corporation. Cambridge, MA. September 27.

Calabrese, E.J. (1993). How valid are EPA's soil ingestion estimates. 8th Annual Soil Contamination Conference. Amherst, MA. September 23.

Calabrese, E.J. (1993). Soil ingestion studies reviewed. Amer. Indust. Health Council. Amherst, MA. September 22.

Calabrese, E.J. (1993). G2 hepatocytes in CCl_4 toxicity. Annual Society of Toxicology. New Orleans, LA.

Calabrese, E.J. (1993). Hepatic ODC activity in fish models. Annual Society of Toxicology. New Orleans, LA.

Calabrese, E.J. (1993). Supercarcinogens. National Center for Toxicological Research (NCTR). Jefferson, Arkansas. September 16.

Calabrese, E.J. (1993). Lead as a mitogen: Effects on CCl_4 hepatotoxicity. NCTR. Jefferson, Arkansas. September 16.

Calabrese, E.J. (1993). G_2 hepatocytes: A new hepatic cellular triage system in response to toxic agents. NCTR. Jefferson, Arkansas. September 16.

Calabrese, E.J. Single exposure carcinogens. (1993). New Jersey Medical School. Newark, NJ. March 3.

Calabrese, E.J. (1993). Toxicological Risk Assessment of DIMP. Colorado Water Quality Control Commission. Denver, CO. March, 1993.

1992

Calabrese, E.J., Leonard, D.A., Baldwin, L.A., Kostecki, P.T. (1992). Ornithine decarboxylase (ODC) activity in the liver of individual medaka (*Oryzias Latipes*). SETAC 13th Annual Meeting. Cincinnati, OH. November 8-12.

Calabrese, E.J., Leonard, D.A., and Baldwin, L.A., (1992). Activated G-2 hepatocytes: A cellular triage system effective against hepatotoxins. SETAC 13th Annual Meeting. Cincinnati, OH. November 8-12.

Bell, C.E., Baldwin, L.A., Kostecki, P.T., and Calabrese, E.J. (1992). Comparative response of rainbow trout and rat to the liver mitogen, lead. SETAC 13th Annual Meeting. Cincinnati, OH. November 8-12.

Calabrese, E.J., Leonard, D.A., and Baldwin, L.A. (1992). Potentiation of CCl4-induced hepatotoxicity by blood drawing. Presented at the SETAC 13th Annual Meeting, November 8-12, Cincinnati, OH.

Calabrese, E.J., Leonard, D.A. and Baldwin, L.A. (1992). Hepatic ornithine decarboxylase (ODC) activity in individual medaka (Oryzias latipes). Annual meeting of the American College of Toxicology, San Francisco, CA. October, 1992.

Calabrese, E.J., Leonard, D.A. and Baldwin, L.A. (1992). Reduction in CCl_4-induced hepatotoxicity by prior treatment with diatomaceous earth. Annual meeting of the American College of Toxicology, San Francisco, CA. October, 1992.

Calabrese, E.J., Leonard, D.A. and Baldwin, L.A. (1992). Reduction in hepatotoxicity by repeated injections of DMN at doses exceeding the MTD. Annual meeting of the American College of Toxicology, San Francisco, CA. October, 1992.

Calabrese, Leonard, D.A. and Baldwin,L.A. (1992). Activated G2 hepatocytes: A cellular triage system effective against hepatotoxins. Annual meeting of the American College of Toxicology, San Francisco, CA. October, 1992.

Calabrese, E.J. (1992). Animal Extrapolation: Future issues. Pfizer, Inc. Groton, CT. September 3, 1992.

Calabrese, E.J. (1992). Chairperson and introductory comments to session on ecological risk assessment. Seventh Annual Hydrocarbon Contaminated Soil Conference, University of Massachusetts, Amherst, MA.

Calabrese, E.J. (1992). Uncertainty factors in ecological risk assessment. Seventh Annual Hydrocarbon Contaminated Soil Conference, University of Massachusetts, Amherst, MA.

Calabrese, E.J. (1992). Effects of peroxisome proliferators on trout and Medaka. U.S. Army Research and Development Lab Annual Research Symposium. Frederick, MD. April 23, 1992.

Calabrese, E.J. (1992). Single exposure carcinogens. Joint EPA, NIEHS Seminar, Research Triangle Park, NC. April 14, 1992.

Calabrese, E.J. (1992). The interdependence of some uncertainty factors: Implications for risk assessment. Conference on New Issues in Occupational Health, Duke University. April 13, 1992.

Calabrese, E.J. (1992). Soil ingestion. Health and Welfare Canada. Toronto, Canada. March 24, 1992.

Calabrese, E.J. (1992). Differentiating soil vs dust ingestion. Third Annual Hydrocarbon Conference. Long Beach, CA. March 12, 1992.

Calabrese, E.J. (1992). Can a single exposure to a chemical carcinogen cause cancer. 3M Corporation, Minneapolis, MN. January 20, 1992.

Calabrese, E.J. (1992). Soil Ingestion in Children. 3M Corporation. Minneapolis, MN. January 20, 1992.

Calabrese, E.J. (1992). Multiple chemical sensitivities. 3M Corporation. Minneapolis, MN. January 20, 1992.

Calabrese, E.J. (1992). Current lead ingestion estimates. Presented at ENSOR Corp., Boston, MA. January 15, 1992.

Calabrese, E.J. (1992). What do we know about soil ingestion? Ensor Corp., Cambridge, MA. January 10, 1992.

1991

Calabrese, E.J. and Kostecki, P. (1991). Soil contaminant research priorities for the 1990's. Department of Engineering, University of Massachusetts. November 23, 1991.

Stewart, J.H., Hosmer, D.W., and Calabrese, E.J. (1991). Estimation and use of the TD50 with the median effect equation in cancer quantitative risk assessment. Society for Risk Analysis. McLean VA. November 16, 1991.

Stewart, J.H., Hosmer, D.W., and Calabrese, E.J. (1991). The median effect equation; its biological plausibility as a model for cancer quantitative risk assessment. Society for Risk Analysis. McLean VA. November 16, 1991.

Calabrese, E.J. (1991). A single dose carcinogens. Health Effects Institute. Cambridge, MA. Oct., 30, 1991.

Calabrese, E.J. (1991). Single doses of carcinogens and cancer risk. Presented at U.S. EPA. Duluth, MN. October 24, 1991.

Calabrese, E.J. (1991). The effects of peroxisome proliferators and mitogens on fish. U.S. EPA. Duluth, MN. October 23, 1991.

Calabrese, E.J., and Stanek, E.J. (1991). Workshop on estimating how much soil children ingest. Presented at the 6th Annual Hydrocarbon Conference. Amherst, MA. September 24, 1991.

Calabrese, E.J. (1991). Soil ingestion estimates: An update presented to the International Lead and Zinc Research Institute. Research Triangle Park, NC. September 9, 1991.

Calabrese, E.J. (1991). A large number of carcinogens can cause cancer with a single dose. ATSDR Guest Seminar. September 6, 1991.

Calabrese, E.J. (1991). How reliable are soil ingestion estimates? ATSDR Guest Seminar. September 6, 1991.

Calabrese, E.J., and Kostecki, P.T. (1991). An update on activities of the council for health and environmental safety of soil (CHESS). ATSDR Guest Seminar. September 6, 1991.

Calabrese, E.J. (1991). Risk communication and public skepticism. U.S. Forest Service, sponsored Malathion Workshop. Arlington, VA. August 27, 1991.

Calabrese, E.J. (1991). Pharmacodynamics/pharmacokinetics of malathion: A discussion of risk assessment models and animal data extrapolation including physiologically-based models in evaluation of malathion human toxicity. U.S. Forest Service sponsored Malathion Workshop. Arlington, VA. August 26, 1991.

Bell, C.E., Kostecki, P.T., and Calabrese, E.J. (1991). Role of risk assessment in state regulatory programs for contaminated soils. Risk-based standards workshop. U.S. Department of Energy. Baltimore, MD. July 9-10, 1991.

Calabrese, E.J. and Stanek, E.J. (1991). Qualitative and quantitative evidence of soil ingestion. Presented at the 15th annual army environmental R & D Symposium. Williamsbury, VA. June 25, 1991.

Calabrese, E.J. (1991). The role pharmacokinetics in facilitating interspecies extrapolation. 7th International Symposium on Radiopharmaceutics. Boston, MA. June 6, 1991.

Calabrese, E.J. (1991). Current issues in risk assessment. Presented at the WHO sponsored course on Toxicology and Risk Assessment. Ottawa, Canada. May 22, 1991.

Calabrese, E.J. (1991). Single exposure carcinogens. Guest seminar for Health and Welfare Canada. Ottawa, Canada. May 22, 1991.

Donohue, M., Baldwin, L., Kostecki, P. and Calabrese, E.J. (1991). The effects of peroxisome proliferators on primary trout hepatocytes. Conference of the American Association on Cancer Research, Houston, Texas. May, 1991.

Scarano, L., Baldwin, L., Kostecki, P., and Calabrese, E.J. (1991). Interactions of peroxisome proliferators in rat. American Association on Cancer Research. Houston, Texas. May, 1991.

Calabrese, E.J. (1991). Short term exposures to potent carcinogens. Invited presentation to the Committee on Toxicology, National Academy of Sciences, Washington, DC. May 15, 1991.

Donahue, M. and Calabrese, E.J. (1991). Peroxisome proliferation in trout. Proceedings of the conference on Regulating Drinking Water in the 1990's. University of Massachusetts, Amherst. April 4, 1990.

Calabrese, E.J. (1991). Uncertainty factors in risk assessment. Presented at Conference on Regulating Drinking Water in the 1990's. University of Massachusetts, Amherst. April 3, 1991.

Gilbert, C.E. and Calabrese, E.J. (1991). Public health risks from SOCs in drinking water. Presented at the Conference on Drinking Water in the 1990's. University of Massachusetts, Amherst. April 3, 1991.

Wysynski, A. and Calabrese, E.J. (1991). Peroxisome proliferators and public health concerns. Presented at the Conference on Regulating Drinking Water in the 1990's. University of Massachusetts. April 3, 1991.

Witko, J. and Calabrese, E.J. (1991). Regulating compliances and SOCs. Proceedings of the conference on Regulating Drinking Water in the 1990's. University of Massachusetts, Amherst. April 3, 1991.

Wysynski, A., Baldwin, L. Kostecki, P. and Calabrese, E.J. (1991). Peroxisomal proliferators: Omega-3 fatty acids, clofibrate and DEHP: the interactive potential. Society of Toxicology. Dallas, February 27, 1991.

Calabrese, E.J. (1991). Chemical carcinogens causing cancer with a single exposure. Implications for risk assessment. University of Oklahoma, School of Public Health. Oklahoma City, Oklahoma. February 27, 1991.

Gilbert, C. and Calabrese, E.J. (1991). Hyperbilirubinemia. A new animal model. Society of Toxicology. Dallas, TX. February, 26, 1991.

Kenyon, E. and Calabrese, E.J. (1991). Interspecies differences in enterohepatic circulation. Society of Toxicology. Dallas, TX. February, 26.

Langlois, C. and Calabrese, E.J. (1991). The interaction of copper, nitrite and chlorite on red blood cells. Presented at the Chemical Oxidation: Technology for the 1990's Conference. Nashville, TN. February 23, 1991.

Calabrese, E.J. (1991). New challenges for risk assessment: What to do about carcinogens causing cancer with a single dose. University of Michigan, School of Public Health. Ann Arbor, MI. February 21, 1991.

1990

Wysynski, A., Baldwin, L., Leonard, D., and Calabrese, E. (1990). The interaction of omega-3 fatty acids with peroxisome proliferators in the rat model. Presented at the New England SOT Regional meeting. Boston, MA. December, 1990.

Kostecki, P. and Calabrese, E.J. (1990). The relevance of CHESS to the oil industry. Presented at an API sponsored meeting in Cleveland, OH. November 20, 1990.

Bell, C.E., P.T. Kostecki and E.J. Calabrese. (1990). UST Cleanup: Concepts, Problems and Alternatives. Paul Smiths College. Paul Smiths, NY. November 13, 1990.

Scarano, L., Baldwin, L., Calabrese, E.J. and Kostecki, P. (1990). Lack of peroxisomal proliferation in Japanese Medaka exposed to DEHP on 2,4-D. Presented at the Society for Environmental Toxicology and Chemistry. Washington, DC. November, 12, 1990.

Yang, J., Calabrese, E.J., Kostecki, P. and Baldwin, L. (1990). Effect of rodent hepatic peroxisomal proliferation on Rainbow Trout. Presented at the Society for Environmental Toxicology and Chemistry. Washington, DC. November 12, 1990.

Calabrese, E.J. (1990). Estimating soil ingestion in children: methodological issues. National Conference on Minority Issues in Environmental Health. Atlanta, GA.

Gilbert, C. and Calabrese, E.J. (1990). Development of a neonatal hyperbilirubinemia rat model for toxicity studies. Presented at the Conference Similarities and Differences Between Children and Adults: Implications for Risk Assessment. Hunt Valley, MN. November 7, 1990.

Gilbert, C. and Calabrese, E.J. (1990). The development of methemoglobin reductase in the neonatal rat. Presented at the Conference on Similarities and Differences Between Children and Adults: Implications for Risk Assessment. Hunt Valley, MN. November 7, 1990.

Kostecki, P., and Calabrese, E.J. (1990). CHESS: An Review of Program. Presented at the 5th Annual Hydrocarbon Contaminated Soil Conference. University of Massachusetts, Amherst. September 26, 1990.

Bell, C., Kostecki, P., and Calabrese, E.J. (1990). Survey of State Approaches for Soil Cleanup Levels. Presented at the 5th Annual Hydrocarbon Contaminated Soil Conference. University of Massachusetts, Amherst. September 26, 1990.

Calabrese, E.J., and Stanek, E.J. (1990). Methodological Advances in Estimating Soil Ingestion. EPA sponsored conference on Lead Exposures to Children. Research Triangle Park, NC. September 24, 1990.

Gilbert, C.E., and Calabrese, E.J., (1990). Educating Youth on the Dangers of Childhood Lead Poisoning. Environmental Health Risk Education for Youth: Curricula Concepts, Strategies and Resources. Interagency Task Force on Environmental Cancer and Lung Disease. September 12-14, 1990. Arlington, VA.

Gilbert, C.E., Jones, T., Calabrese, E.J., and Winder, A. (1990). Environmental Curricula Concerning Waste Management. Environmental Health Risk Education for Youth: Curricula Concepts, Strategies and Resources. Interagency Task Force on Environmental Cancer and Lung Disease. September 12-14, 1990. Arlington, VA.

Langlois, C., Leonard, D., and Calabrese, E.J. (1990). Interactions of multiple methemoglobin-forming agents. New England SOT Regional Meeting, Boston. June 1, 1990.

Gilbert, C., and Calabrese, E.J. (1990). Development of a neonatal hyperbilirubinemia model. Maine Biological Symposium, Mt. Dessert Island. May 30, 1990.

Stewart, J., and Calabrese. (1990). The median effect principle in toxicology and risk assessment. Maine Biological Symposium, Mt. Dessert Island. May 30, 1990.

Calabrese, E.J. (1990). A toxicological appraisal drinking water disinfectants and implications for risk assessment. National Conference on Drinking Water and Health, Amherst, MA. April 30, 1990-May 2, 1990.

Gilbert, C., and Calabrese, E.J. (1990). MTBE-a critical evaluation of its toxicological data base. National Conference on Drinking Water and Health, Amherst, MA. April 30, 1990-May 2, 1990.

Langlois, C., Leonard, D., and Calabrese, E.J. (1990). The effects of multiple exposure of the drinking water oxidants, chlorite, nitrite and copper on red blood cells. National Conference on Drinking Water and Health, Amherst, MA. April 30, 1990-May 2, 1990.

Stewart, J., and Calabrese, E.J. (1990). The application of the median effect principle for assessing risk to drinking water contaminants. National Conference on Drinking Water and Health, Amherst, MA. April 30, 1990-May 2, 1990.

Calabrese, E.J. (1990). Acute toxicities and cancer risks: the problem of single exposures. Presented at the U.S. Environmental Protection Agency, Washington, DC. April 11, 1990.

Calabrese, E.J. (1990). A single exposure to a carcinogen can cause cancer. Presented at the Center for Environmental Toxicology, Michigan State University, East Lansing, MI. April 3, 1990.

Calabrese, E.J. (1990). Single exposures and cancer risks. Presented to the participants of the EPA sponsored workshop on Acute Toxicities. Washington, DC. March 12, 1990.

Bell, C., Kostecki, P. and Calabrese, E.J. (1990). Petroleum contaminated soils survey: clean-up levels for western states. Presented at conference on Hydrocarbon Contaminated Soils and Groundwater. Newport Beach, CA. February 19-22, 1990.

Kostecki, P. and Calabrese, E.J. (1990). Council for Health and Environmental Safety of Soils-CHESS. Presented at conference on Hydrocarbon Contaminated Soils and Groundwater. Newport Beach, CA. February 19-22, 1990.

Edmisten, G., Calabrese, E.J. and Harris, P. (1990). Health risks associated with the remediation of contaminated soils. Presented at conference on Hydrocarbon Contaminated Soils and Groundwater. Newport Beach, CA. February 19-22, 1990.

Calabrese, E. (1990). Methodological approaches for assessing soil ingestion. Presented at conference on Hydrocarbon Contaminated Soils and Groundwater. Newport Beach, CA. February 19-22, 1990.

Calabrese, E.J. (1990). Methodological approaches to assessing chemical interactions of toxicological significance. Presented at the Aberdeen Proving Ground, Maryland, Department of Defense. June 24, 1990.

Calabrese, E.J. (1990). Soil ingestion in children. Environ Corp., Princeton, NJ. January 26, 1990.

1989

Calabrese, E.J. (1989). Interspecies variations in enterohepatic recirculation of PCBs and the implications for cancer risk. General Electric Sponsored Research Seminar. Arlington, VA. November 29, 1989.

Kostecki, E.J. (1989). CHESS: Its role in assessing soil cleanup levels. Dept. of Defense Environ. Conference. Williamsburg, VA. (November 16, 1989.

Calabrese, E.J. (1989). Can a single exposure to a carcinogen cause cancer. Presented at the Chemical Defense Research Conference. Auberdeen, MD. November 14, 1989.

Bell, C., Kostecki, P. and Calabrese, E.J. (1989). Survey of state regulatory programs for soil clean-up. EPA sponsored conference on state regulatory programs for underground storage tanks. Alberque, New Mexico. November 11, 1989.

Calabrese, E.J. (1989). The health effects of DIMP. Colorado Department of Health. Denver, CO. November 8, 1989.

Calabrese, E.J. (1989). One exposure study and chemical carcinogenesis. Seminar, Department of Environmental Engineering, University of Massachusetts, Amherst, MA. October 13, 1989.

Ochs, J., Calabrese, E.J. et al. (1989). The joint exposure of two peroxisome proliferation agents on hepatic fatty acid oxidase activity in mice. Presented at New England Regional Chapter of the Society of toxicology. Sturbridge, MA. October 20, 1989.

Scarano, G., Calabrese, E.J. et al. (1989). The capacity of Rainbow Trout to display hepatic peroxisome proliferation. Presented at New England Regional Chapter of the Society of toxicology. Sturbridge, MA. October 20, 1989.

Nolan, K. and Calabrese, E.J. (1989). The effect of vitamin C on intestinal, cecal, and urinary B-glucuronidase activity in the rodent. Presented at New England Regional Chapter of the Society of toxicology. Sturbridge, MA. October 20, 1989.

Calabrese, E.J. and Sonich-Mullin C. (1989). Genetic factors and susceptibility to occupational illness. Presented at WHO Conference, Drefeld Federal Republic of Germany. October 17-20, 1989.

Calabrese, E.J. et al. (1989). Results of a pilot study to estimate soil ingestion in adults. In: National Conference on Petroleum Contaminated Soils Conference. University of Massachusetts, Amherst, MA. September 25-29, 1989.

Stanek, E.J., Calabrese, E.J. et al. (1989). Improved estimates of soil ingestion in children. In: National Conference on Petroleum Contaminated Soils Conference. University of Massachusetts, Amherst, MA. September 25-29, 1989.

Bell, C., Kostecki, P. and Calabrese, E.J. (1989). National survey of regulatory approaches to remediation of petroleum contaminated soils. In: National Conference on Petroleum Contaminated Soils Conference. University of Massachusetts, Amherst, MA. September 25-29, 1989.

Gilbert, C. and Calabrese, E.J. (1989). Methodological approaches for selecting indicator compounds for home heat fuel number 2. In: National Conference on Petroleum Contaminated Soils Conference. University of Massachusetts, Amherst, MA. September 25-29, 1989.

Calabrese, E.J. (1989). Toxicological Risk Assessment of DIMP. Colorado Water Quality Control Commission. Denver, CO. September, 1989.

Calabrese, E.J. (1989). Peroxisome proliferation in fish. Annual Aquatic Toxicology Research meeting. Department of Defense. Ft. Detrick, Maryland. August, 9, 1989.

Calabrese, E.J. (1989). Toxicological Risk Assessment of DIMP. Colorado Water Quality Control Commission. Denver, CO. July,1989.

Calabrese, E.J. (1989). The role of toxicology in assessing risks for naturally occurring toxins in the food supply. Food and Nutrition Board of the National Academy of Sciences. Falmouth, MA. July, 23, 1989.

Bell, C., Kostecki, P. and Calabrese, E.J. (1989). State approaches for the clean-up of petroleum contaminated soil. Maine Biological Science Conference, Portland, Maine. June 3, 1987.

Calabrese, E.J., and Kostecki, P. (1989). Biomarkers for toxicology studies in fish. Procter and Gamble, Cincinnati. June 12, 1989.

Kostecki, P., and Calabrese, P. (1989). International approaches for assessing health risks from contaminated soils. National Public Health Association Conference, San Antonio, Texas. June 21, 1989.

Calabrese, E.J. (1989). Peroxisomes proliferation, carcinogenesis, and implications for risk assessment. Annual Conference on Aquatic Toxicology, ASTM, Atlanta, Georgia. April 18, 1989.

Calabrese, E.J. (1989). Single exposures to chemical carcinogens can cause cancer. Agency for Toxic Substances and Disease Registry, Atlanta, Georgia. April, 1989.

Calabrese, E.J. (1989). Assessing cancer risk when a single exposure to a carcinogen causes cancer. Amer-Indus. Health Council, Washington, DC. April, 1989.

Calabrese, E.J. (1989). Predicting toxicological responses from multiple chemical exposures. University of Illinois, Champaigne/Urbana, Illinois. April 5, 1989.

Calabrese, E.J. (1989). Less than lifetime exposure to carcinogens and risk assessment methodologies. Dartmouth Medical School, Hanover, NH.

Calabrese, E.J. (1989). Public health concerns of medical waste disposal. Sponsored by the Rockefeller Institute of Government. New York, NY. March 9, 1989.

Calabrese, E.J. (1989). Genetic susceptibility to occupationally-induced disease. Regional chapter of the American Industrial Hygiene Association (Conn. and NY). Stanford, CT. February 14, 1989.

Kostecki, E.J., and Calabrese, E.J. (1989). Leaking underground storage tanks and public health concerns. Annual New England Water Pollution Control Assoc., Boston, MA. (Jan. 23, 1989).

Calabrese, E.J. (1989). The role of genetic screening in the prevention of occupationally-induced disease. Johns Hopkins University, Baltimore, Maryland. January 9, 1989.

1988

Bell, C.E., Calabrese, E.J., Kostecki, P.T. (1988). State of research and regulatory approach of state agencies for cleanup of petroleum contaminated soils. Presented at the First Annual Real Estate Site Assessment Conference, Resource Education Institute. Sturbridge, Massachusetts. December 1988.

Kenyon, E., and Calabrese, E.J. (1988). Inter-species differences in gastrointestinal B-glucuronidase activity. New England Mutagen Society, Kingston, Rhode Island. October 1988.

Kostecki, P., and Calabrese, E.J. (1988). Peroxisome proliferation in fish. New England Mutagen Society. Kingston, Rhode Island. October 1988.

Yang, J., Calabrese, E.J., and Kostecki, P. (1988). Peroxisome proliferation in the rainbow trout. New England Chapter of the Society of Toxicology. Boston, Massachusetts. October 1988.

Bell, C.E., Kostecki, P. and Calabrese, E.J. (1988). National survey of state approaches for regulating petroleum contaminated soil. Third Conference on Environmental and Public Health Effects of Soils Contaminated with Petroleum. Amherst, MA. September 19-21, 1988.

Calabrese, E.J. (1988). Determining the health hazard associated with complex mixtures such a petroleum products. Third Conference on Environmental and Public Health Effects of Soils Contaminated with Petroleum. Amherst, MA. September 19-21, 1988.

Calabrese, E.J. et al. (1988). Soil ingestion in children. Third Conference on Environmental and Public Health Effects of Soils Contaminated with Petroleum. Amherst, MA. September 19-21, 1988.

Kostecki, P. and Calabrese, E.J. (1988). Council for the Health and Environmental Safety of Soils (CHESS). Third Conference on Environmental and Public Health Effects of Soils Contaminated with Petroleum. Amherst, MA. September 19-21, 1988.

Calabrese, E.J. (1988). Soil ingestion and implications for risk assessment. Annual Risk Assessment Conference sponsored by the Center for Energy and Environmental Management. Alexandria, Virginia.

Calabrese, E.J. (1988). Peroxisome proliferation in fish. U.S. Army Biomedical Corp. Annual Meeting. Fort Detrick, MD. August 23, 1988.

Calabrese, E.J. (1988). Air toxic - a new methodology. Chemical Manufacturers Association sponsored conference on Community Exposures. Boston, MA. August 17, 1988.

Calabrese, E.J. (1988). The problem of soil ingestion by children. Annual EPA Risk Assessment Conference. Philadelphia, PA. June 27, 1988.

Calabrese, E.J. (1988). Exposure quantification: soil ingestion. Future Technologies Conference. Clark Univ./WPI. Worcester, MA. June 15, 1988.

Calabrese, E.J. (1988). Recent epidemiological evidence of soil ingestion by children. Univ. Michigan, Ann Arbor, MI. June 13, 1988.

Calabrese, E.J. (1988). Soil ingestion by children. American Industrial Health Assoc. Washington, DC. June 9, 1988.

Calabrese, E.J. (1988). Principles of animal extrapolation and their application. Amer. Chem. Society. Short Course on Toxicology. Clearwater, Florida. June 3, 1988.

Calabrese, E.J. (1988). Estimating Soil Ingestion in Children. Agency for Toxic Substances and Disease Prevention. Atlanta, Georgia. June 2, 1988.

Coler, R., Kostecki, P. and Calabrese, E.J. (1988). Assessment of the effect of chlorination practices on selected aquatic communities. Northeast Regional Environ. Conference. Amherst, MA. May 28, 1988.

Kostecki, P., Calabrese, E.J. and Coler, R. (1988). The aquatic toxicology program of the Massachusetts Fisheries and Wildlife Department. Regional Environ. Conference. Amherst, MA. May 28, 1988.

Calabrese, E.J. (1988). Municipal solid waste disposal - Introductory Remarks. Conference sponsored by the Northeast Regional Environmental Public Health Center. April 19, 1988. Amherst, MA.

Calabrese, E.J. (1988). Estimating soil ingestion in children. EPA Special Colliquium. Washington, DC. March 23, 1988.

Calabrese, E.J. (1988). Sodium: A Changing Public Health Perspective? Annual Meeting of the American R. Water Association. Reno, NV. March 19, 1988.

Kostecki, P.K. and Calabrese, E.J. (1988). Developing a consistent approach for assessing public health risks from contaminated soil. American Conference of Governmental Industrial Hygienist sponsored conference, at Arlington, VA. March 1, 1988.

Gilbert, C.E. and Calabrese, E.J. (1988). Regional approaches for risk management. National Conference of the Mosquito Control Association. Denver, CO. February 23, 1988.

Calabrese, E.J. (1988). Soil ingestion in children: Methodological approaches. Mobil Oil Company. Princeton, NJ. January 7, 1988.

1987

Calabrese, E.J. (1987). Recent advances in animal extrapolation. Presented at the Agency for Toxic Substances and Disease Registry. Atlanta, GA. December 4, 1987.

Calabrese, E.J. (1987). Estimates of soil ingestion in children: A proposed methodology. U.S. Public Health Service Conference. Hyannis, MA. December 1, 1987.

Calabrese, E.J. et al. (1987). Reproductive health outcome study at a DEC facility. National Conference on Semi-Conductor Health. Cincinnati, OH. Oct. 21, 1987.

Calabrese, E.J. (1987). A model air toxins program. Rohm & Haas, Inc. Phil. Oct. 15, 1987.

Calabrese, E.J. (1987). Introductory and chairman remarks on session on inhalation toxicology at conference on Animal Extrapolation. Duke University, N.C. Oct. 9, 1987.

Calabrese, E.J. (1987). Report on the health assessment of drinking treatment technologies. Environmental Scientific Advisory Board (SAB), Washington, D.C. Oct. 8, 1987.

Calabrese, E.J. (1987). Reproductive hazards in the semi-conductor industry. National Safety Council Annual Meeting, Chicago. (Oct. 5, 1987).

Kostecki, P., Horton, H.M. and Calabrese, E.J. (1987). Comparison of models to protect health effects from soil contamination. Second Conference on Environmental and Public Health Effects of Petroleum Contaminated Soils. Amherst, MA. September 30, 1987.

Calabrese, E.J. (1987). Epidemiologic study to estimate soil ingestion in children. Second Conference on Environmental and Public Health Effects of Petroleum Contaminated Soils. Amherst, MA. September 29, 1987.

Calabrese, E.J. (1987). Predictive Toxicology. American Chemical Society Meeting. Cincinnati. June 18.

Calabrese, E.J. (1987). The toxicologist and risk communication. Conference on Environmental Risk Communication. Amherst, Massachusetts. June 9.

Yang, J. and Calabrese, E.J. (1987). Studies on the in vitro capacity of ethanol to enhance sodium nitrite and l-naphthol-induced oxidant stress in human and sheep erythrocytes. Biomedical Science Conference, Bowdoin College, Maine. June 4.

Tilli, F. and Calabrese, E.J. (1987). The effect of ethanol on the response of normal human erythrocytes to 12 oxidant stressors. Biomedical Science Conference. Bowdoin College, Maine. June 4.

Kenyon, E.M., Young, J., and Calabrese, E.J. (1987). Inhibition of B-glucuronidase in human urine by ascorbic acid. Biomedical Science Conference. Bowdoin College, Maine. June 3.

Kenyon, E.M. and Calabrese, E.J. (1987). B-glucuronidation activity in the small intestine of mice, rats and rabbits. Biomedical Science Conference. Bowdoin College, Maine. June 3.

Calabrese, E.J. (1987). Public health concerns and high technology. Conference on Technology and Public Health, Worcester, Massachusetts. May 28.

Calabrese, E.J. (1987). Conference Summary on Ozone Toxicology. Ozone Risk Communication Conference. Amherst, Massachusetts. April 22.

Fleischer, E. and Calabrese, E.J. (1987). Soil Venting and Public Health Risks. Soil Remediation/Technology Conference. Sturbridge, Massachusetts. April 6.

Kostecki, P.T. and Calabrese, E.J. (1987). Petroleum Contamination of Soils - State of the art for environmental and public health assessment. Soil Remediation/Technology Conference. Sturbridge, Massachusetts. April 6.

Calabrese, E.J. (1987). Toxicology and Drinking Water Regulations. N.E. AWWA Meeting. Windsor Locks, Connecticut. March 19.

Calabrese, E.J. (1987). Principles of Animal Extrapolation U.S.D.A. Toxicology and Risk Assessment Conference. Atlanta Georgia. February 11.

Calabrese, E.J. (1987). Asbestos in Play Sand? Introductory remarks to conference. Conference on Asbestos in Play Sand. Northeast Regional Environmental Public Health Center, University of Massachusetts, Amherst. February 10.

Calabrese, E.J., Pastides, H. and Hosmer, D. (1987). Health surveillance assessment in the semi conductor industry. Windsor Locks, Connecticut. January 23.

Calabrese, E.J. (1987). Health concerns from groundwater contaminants. Third National Drinking Water Conference. Philadelphia, Pennsylvania. January 13, 1987.

1986

Calabrese, E.J. (1986). Predictive Toxicology. U.S. Army, Fort Detrick, Maryland. December 14, 1986.

Calabrese, E.J. (1986). Advances in animal extrapolation. Regional Meeting of the Halogenated Solvents Industry Alliance Atlanta, Georgia. December 10, 1986.

Kostecki, P. and Calabrese, E.J. (1986). A review of formal and informal soil standards within the U.S. Amer. Soc. of Agronomy. 78th Annual Meeting, New Orleans, Louisiana. December 4, 1986.

Calabrese, E.J. (1986). Regional approaches for addressing environmental health concerns. U.S. Public Health Service Region 1, Annual Conference. Hyannis, Massachusetts. December 3, 1986.

Calabrese, E.J. (1986). Chemical interactions in environmental health. American College of Toxicology Annual Meeting. November 17, 1986. Philadelphia, Pennsylvania.

Calabrese, E.J. (1986). Chairman - Session on Drug/Chemical Interactions. American College of Toxicology Annual Meeting. November, 17, 1986.

Calabrese, E.J. (1986). Predictive toxicology; principles and applications. Amer. Chem. Soc. Ann. Meeting. November 14, 1986, Florida.

Kostecki, P.T., Calabrese, E.J. and Garnick, E. (1986). A national census of how petroleum contamination is being assessed. Soc. Environ. Chem. and Tox. November 5, 1986. Washington, D.C.

Calabrese, E.J. (1986). Principles of animal extrapolation and their application for pesticide risk assessment. Soc. Environ. Chem. and Tox. November 5, 1986, Washington, DC.

Kostecki, P.T. and Calabrese, E.J. (1986). The importance of environmental factors on the reproductive success of smelt (Osmerus mordax). Soc. Environ. Chem. Tox. November 4, 1986, Washington, D.C.

Calabrese, E.J. (1986). The effect of vitamin C supplementation of the body burden of heavy metals. Third International Conference on Vitamin C. October 6, 1986. New York.

Calabrese, E.J. (1986). Inhibitor of urinary B-glucuronidase activity in human subjects by vitamin C supplementation. Third International Conference on Vitamin C. October 6, 1986. New York.

Calabrese, E.J. (1986). The toxicological basis for establishing National Primary Drinking Water Standards. Conference on the Safe Drinking Water Act. September, 23, 1986. Amherst, Massachusetts.

Calabrese, E.J. (1986). Regional approaches for environmental public health policy. Annual meeting of the New England Interstate Water Commission, Kennebunkport, Maine. September 9, 1986.

Calabrese, E.J. (1986). Assessing the effects of toxic substances. The Samuel Johnson Memorial Lecture. Connecticut Agricultural Research Station. August 6, 1986. New Haven, Connecticut.

Calabrese, E.J. (1986). Inhibition of B-glucuronidase activity and susceptibility to cancer. Proctor and Gamble. June 30, 1986. Cincinnati, Ohio.

Calabrese, E.J. (1986). Role of academia in reducing exposure to toxic substances. National Environmental Health Association. June 16, 1986. Hartford, Connecticut.

Calabrese, E.J. (1986). Regional approaches for assessing environmental contamination. New England Laboratory Directors Quarterly Meeting. June 3, 1986. Oqunquit, Maine.

Calabrese, E.J., and McCarthy, M.E. (1986). The occurrence of trace-metal induced hormesis. 20th Annual Conference on Trace Substances in Environmental Health June 2-5, 1986. University of Missouri, Columbia.

Calabrese, E.J., and Kostecki, P. (1986). Approaches for assessing the public health significance of soil contaminated with toxic agent. 20th Annual Conference on Trace Substances in Environmental Health. June 2-5, 1986. University of Missouri, Columbia.

Calabrese, E.J. and McCarthy, M.E. (1986). The occurrence of trace-metal induced hormesis. Presented at the Maine Biological and Medical Sciences Symposium. May 29, 1986, Portland, Maine.

Byrne, K., Kostecki, P.T., and Calabrese, E.J. (1986). The importance of environmental factors on reproductive success of smelt. Presented at the Maine Biological and Medical Sciences Symposium. May 29, 1986, Portland, Maine.

Kostecki, P., Calabrese, E.J., and Garnick, E. (1986). A national survey of regulatory approaches for addressing soil contaminated with petroleum products. Presented at the Maine Biological and Medical Science Symposium. May 28, 1986. Portland, Maine.

Calabrese, E.J. (1986). Animal extrapolation and the problem of human interindividual variation. Presented to the New England Mutagenesis Society. May 16, 1986. Amherst, Massachusetts.

Calabrese, E.J. (1986). Age and susceptibility to pollutant induced toxicity. National Academy of Science. May 15, 1986. Washington, D.C.

Calabrese, E.J. (1986). Predicting human health risks from exposure to contaminated soil. Presented at the EPA-sponsored Conference. May 8, 1986, Andover, Massachusetts.

Calabrese, E.J. (1986). Animal extrapolation and its regulatory implications. Presented to the Air Pollution Control Association. April 22, 1986, Providence, Rhode Island.

Calabrese, E.J. (1986). Principles of inter-, intra-species extrapolation. Presented to the Society of Risk Analysis. April 9, 1986, Washington, D.C.

Calabrese, E.J. (1986). The effects of toxic substances on males and females. Annual Digital Equipment Corporation Conference. April, 1986. Merrimack, New Hampshire.

Calabrese, E.J. (1986). Risk assessment methodologies: Strengths and limitations. Annual Digital Equipment Corporation Conference. April, 1986. Merrimack, New Hampshire.

Canada, A.T., Chow, C.K., and Calabrese, E.J. (1986). Effect of O3 on serum concentrations of vitamins A, C and E in mature female rabbits. Presented to the Annual Meeting of the Society of Toxicology. March 1986, New Orleans, Louisiana.

Calabrese, E.J. (1986). How relevant is the rat? presented to the American Industrial Hygiene Association. Meridan, Connecticut.

Calabrese, E.J. (1986). Interspecies differences in xenobiotic metabolism. Presented to the American Pharmaceutical Association. March 18, 1986, San Francisco, California.

Calabrese, E.J. (1986). New approaches for animal extrapolation. Presented to the Electric Power Research Institute. March 17, 1986, Palo Alto, California.

Calabrese, E.J. (1986). Regional strategies for assessing risk from environmental toxins. Presented to the State of Connecticut's Department of Environmental Analysis. January 11, 1986, New Haven, Connecticut.

Calabrese, E.J. (1986). Animal extrapolation and the challenge of human heterogeneity. Presented to an FDA-sponsored conference. January 6, 1986, Bethesda, Maryland.

1985

Calabrese, E.J. (1985). Regional approaches for addressing environmental problems. Presented to the CDC Annual Region 1 Conference. December 1985, Hyannis, Massachusetts.

Kostecki, P.T. and Calabrese, E.J. (1985). Environmental and public health effects of petroleum contaminated soils. Presented at the Annual Meeting of the American College of Toxicology. November 1985, Amherst, Massachusetts.

Kostecki, P.T. and Calabrese, E.J. (1985). Problems associated with petroleum contaminated soils. Presented at the Annual Meeting of the Society of Environmental Toxicology and Chemistry. November 1985, St. Louis, Missouri.

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XIV. BOOKS

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XV. CONFERENCE PROCEEDINGS - EDITORSHIP

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36. An EPA-sponsored international conference on The Influence of Drinking Water on the Occurrence of Cardiovascular Disease. Published within the Journal of Environmental Pathology and Toxicology. 4:1-326, 1981.

37. An EPA-sponsored international conference on The Effects of Pollutants on Human High Risk Groups. Published within Environmental Health Perspectives. 29:1-77, 1979.

XVI. NEWSPAPER COLUMNIST

I wrote a bimonthly column for the *Amherst Record* on general topics in environmental health from May 1982-1985. On occasion, I write a guest column for other newspapers including the *Hartford Courant*.

XVII. NEWSLETTER

1. Health Effects Section Writer - for the 4500 members of the American Water Works Association, 1982/1983.

2. Biological Effects of Low Level Exposures (BELLE) Newsletter. A publication of the Northeast Regional Environmental Public Health Center, University of Massachusetts, School of Public Health. 11,000 circulation; 1992 - 2010.

3. Council for Health and Environmental Safety of Soils – CHESS Newsletter. Published by the International Society of Regulatory Toxicology and Pharmacology. 1989-1993.